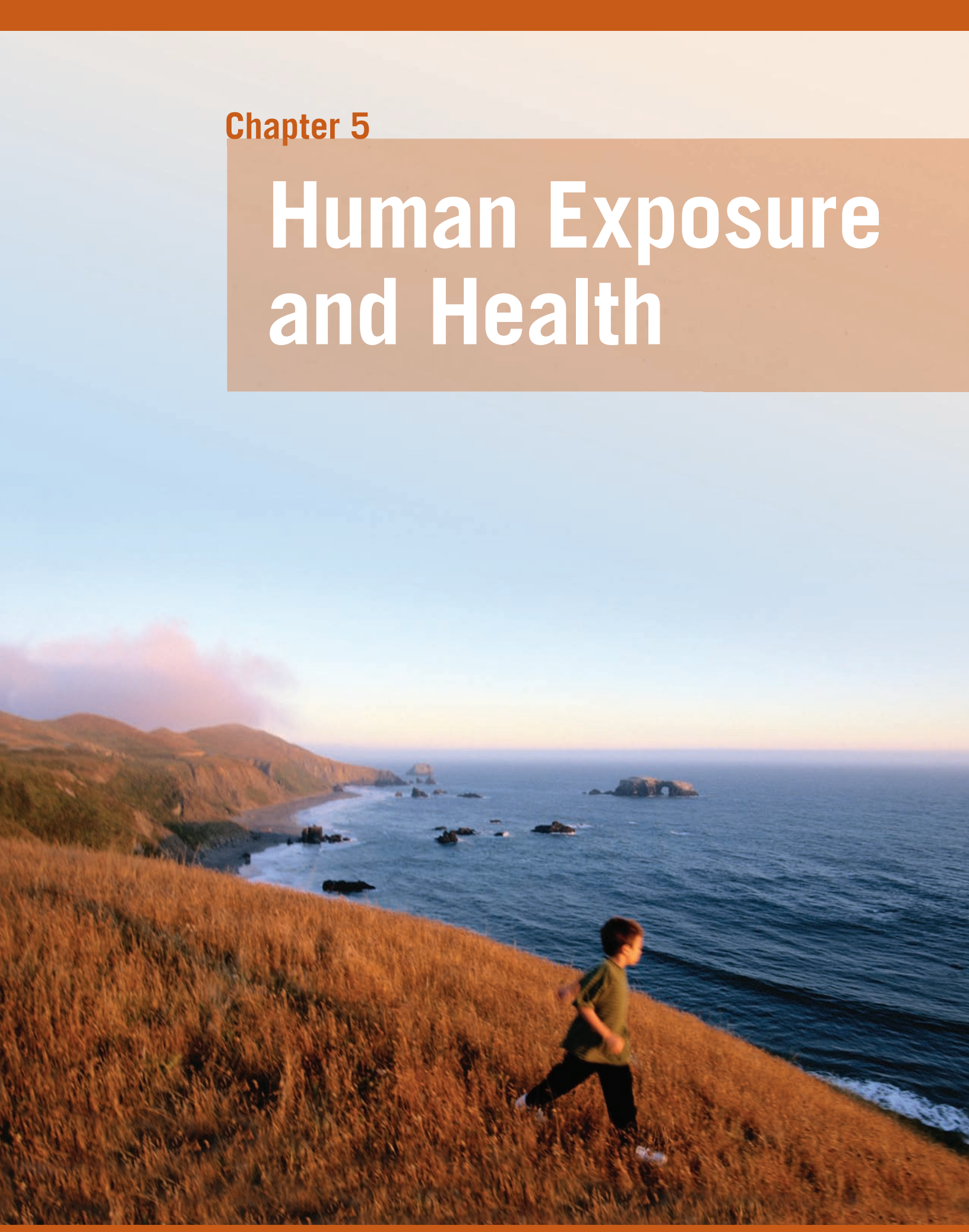
Chapter 5

Human Exposure and Health



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5.1 Introduction

The health of the human population can be influenced by many factors, one of which is exposure to environmental contamination. Protecting human health from the effects of environmental contaminants is therefore an integral part of EPA's mission. Protecting, sustaining, or restoring the health of people and communities is central to EPA's various research and regulatory programs. In fulfilling its mission, EPA examines the human health impacts of contamination (physical, chemical, biological, or radiological) in air, in water, and on the land. Thorough study of adverse health effects associated with environmental contaminants enables the Agency to evaluate harmful levels of exposure and issue guidelines for the safe production, handling, and management of hazardous substances.

As described in Chapters 2 through 4, people can be exposed to environmental contaminants in a variety of ways, and many contaminants are known to be or suspected of causing human disease. Identifying (1) the extent to which human exposures may be occurring or may have occurred and (2) measures of health outcomes possibly influenced by environmental exposures is important in determining where further study or public health interventions may be necessary. For example, the presence or patterns of elevated levels of environmental contaminants, as measured in human tissue through biomonitoring, is of interest. Similarly, a high or increasing rate of a particular cancer for which a hazardous substance in the environment may be a contributing factor is of interest. In addition, tracking exposures and health condition across segments of the population such as gender, race or ethnicity, or geographic location helps to identify differences across subgroups and guide public health decisions and strategies.

In this chapter, EPA seeks to assess trends in human exposure and disease or conditions that may be associated with environmental factors on a national scale. Biomonitoring and health outcome indicators are presented to address three fundamental questions:

- **What are the trends in human exposure to environmental contaminants, including across population subgroups and geographic regions?** Data on trends in exposure levels provide an opportunity to evaluate the extent to which environmental contaminants are present in human tissue, independent of the occurrence of specific diseases or conditions. To address this question, this chapter focuses on biomonitoring indicators (or biomarkers of exposure) for environmental contaminants such as lead, mercury, and pesticides.
- **What are the trends in health status in the United States?** Here the report uses several general health outcome indicators (life expectancy, infant mortality, and general mortality) to provide a broad picture of health in the U.S. Trends in these indicators provide a general context for understanding trends in specific diseases and conditions that may in part be linked with the environment.
- **What are the trends in human disease and conditions for which environmental contaminants may be a risk factor, including across population subgroups and geographic regions?** This question looks at the occurrence of diseases and conditions that are known that are influenced by many stressors acting through multiple media and by factors outside EPA's mission.

EPA's 2008 Report on the Environment (ROE): Essentials

ROE Approach

This 2008 Report on the Environment:

- Asks questions that EPA considers important to its mission to protect human health and the environment.
- Answers these questions, to the extent possible, with available indicators.
- Discusses critical indicator gaps, limitations, and challenges that prevent the questions from being fully answered.

ROE Questions

The air, water, and land chapters (Chapters 2, 3, and 4) ask questions about trends in the condition and/or extent of the environmental medium; trends in stressors to the medium; and resulting trends in the effects of the contaminants in that medium on human exposure, human health, and the condition of ecological systems.

The human exposure and health and ecological condition chapters (Chapters 5 and 6) ask questions about trends in aspects of health and the environment

ROE Indicators

An indicator is derived from actual measurements of a pressure, state or ambient condition, exposure, or human health or ecological condition over a specified geographic domain. This excludes indicators such as administrative, socioeconomic, and efficiency indicators.

Indicators based on one-time studies are included only if they were designed to serve as baselines for future trend monitoring.

All ROE indicators passed an independent peer review against six criteria to ensure that they are useful; objective; transparent; and based on data that are high-quality, comparable, and representative across space and time.

Most ROE indicators are reported at the national level. Some national indicators also report trends by region. EPA Regions were used, where possible, for consistency and because they play an important role in how EPA implements its environmental protection efforts.

Several other ROE indicators describe trends in particular regions as examples of how regional indicators might be included in future versions of the ROE. They are not intended to be representative of trends in other regions or the entire nation.

EPA will periodically update and revise the ROE indicators and add new indicators as supporting data become available. In the future, indicators will include information about the statistical confidence of status and trends. Updates will be posted electronically at http://www.epa.gov/roe.

Additional Information

You can find additional information about the indicators, including the underlying data, metadata, references, and peer review, at http://www.epa.gov/roe.

or suspected to be caused (to some degree) or exacerbated by exposures to environmental contaminants. This chapter uses a spectrum of indicators for health outcomes—such as cancer, asthma, and birth outcomes—to address this question. Both morbidity and mortality statistics are considered.

These ROE questions are posed without regard to whether indicators are available to answer them. This chapter presents the indicators available to answer these questions, and also points out important gaps where nationally representative data are lacking.

This chapter is not intended to be exhaustive in addressing these questions, nor is it intended to be a risk assessment or epidemiological study. Rather, it provides an overview of selected indicators of human exposure and disease over space and time, based on key data sources with sufficiently robust design and quality assurance.

The indicators used here are based on data sets representative of the national population; they are not based on data from targeted populations or tied to specific exposures or releases. Therefore, these data sets cannot and should not be used to draw conclusions about linkages or causal relationships between a particular health outcome and contaminant; nor is it possible to directly link the health outcome or biomonitoring indicators to any of the indicators of emissions or ambient pollutants in air, land, or water presented in earlier chapters of this report. Though the chapter does not assess quantitative relationships between the measures of environmental contaminants and diseases, it does present some qualitative discussion of the research that has examined some of these relationships to help explain why EPA has included particular indicators. Sections 5.1.1 and 5.1.2 detail important principles guiding the selection and interpretation of exposure and health indicators used in this report.

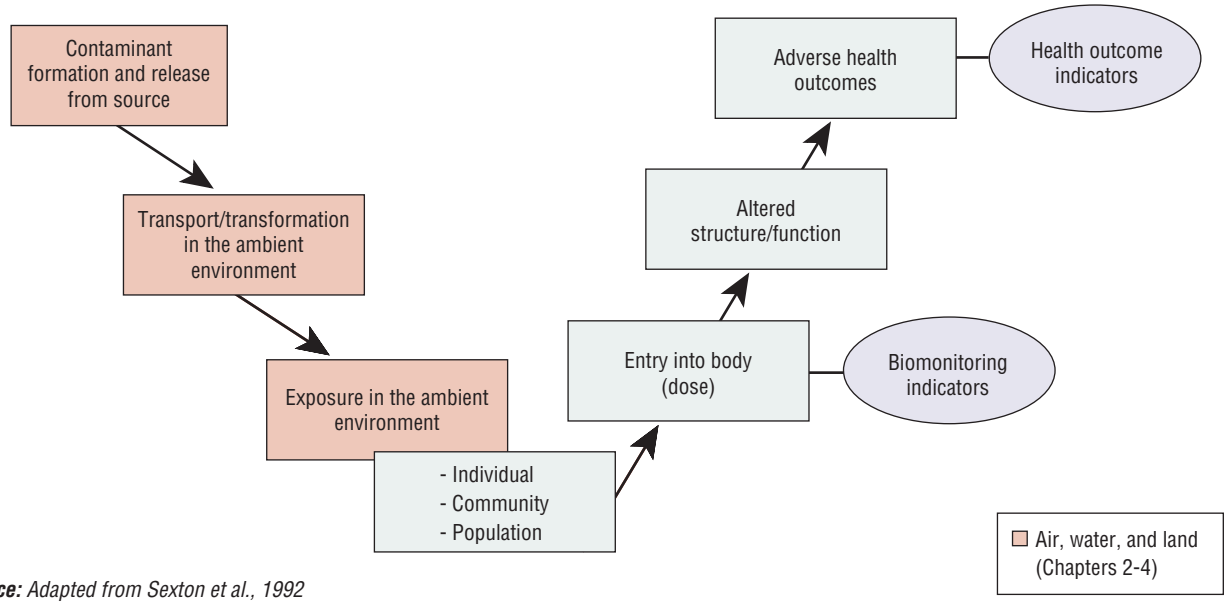
5.1.1 The Environmental Public Health Paradigm

The relationship among and between environmental contamination, exposure, and disease is complex. Development of disease is multi-faceted. Relationships between environmental exposures and various health outcomes can only be established through well-designed epidemiological, toxicological, and clinical studies. An understanding of these factors provides critical context for this chapter.

The environmental public health paradigm shown in Exhibit 5-1[1] illustrates the broad continuum of factors or events that may be involved in the potential development of human disease following exposure to an environmental contaminant. This series of events serves as the conceptual basis for understanding and evaluating environmental health. The exhibit illustrates that for adverse health effects (clinical disease or death) to occur, many things have to happen. A contaminant must be released from its source, reach human receptors (via air, water, or land), enter the human body (via inhalation, ingestion, or skin contact), and be present within the body at sufficient doses within individuals to cause biological changes that may ultimately result in an observed adverse health effect.

The paradigm, however, is a linear, schematic depiction of a process that is complex and multi-factorial. Exposure to an environmental contaminant is rarely the sole cause of an adverse

Exhibit 5-1. Environmental public health paradigm



Source: *Adapted from Sexton et al., 1992*

[1] Adapted from: Sexton, K., S.G. Selevan, D.K. Wagener, and J.A. Lybarger. 1992. Estimating human exposures to environmental pollutants: Availability and utility of existing databases. Arch. Environ. Health 47(6):398-407.

health outcome. Environmental contaminant exposure is just one of several factors that can contribute to disease occurrence or to the severity of a preexisting disease. Among the other factors are diet, exercise, alcohol consumption, individual genetic makeup, medications, and other pre-existing diseases. Asthma, for example, can be triggered by environmental insult, but environmental exposures are not the "cause" of all asthma attacks. In addition, different contaminants can be a risk factor for the same disease. Taking the same example, outdoor air pollution and certain indoor air pollutants, such as environmental tobacco smoke, can both exacerbate asthma symptoms. Further, susceptibility to disease is different for each person; some individuals may experience effects from certain ambient exposure levels while others may not.

Each block in Exhibit 5-1 can have indicators associated with it. As shown, aspects of Chapters 2 through 4 may address contaminant formation, release, transport, and transformation in the environment. Those chapters present indicators for the presence of contaminants or other stressors affecting air, water, and land, sometimes at locations in which people may be exposed. Measurements of ambient exposure levels are different than the biomonitoring indicators (biomarkers of exposures) introduced in this chapter. Other types of biomarkers exist (e.g., biomarkers of susceptibility and biomarkers of effect); because national-scale data do not exist for these biomarkers, they are not covered in this chapter at this time.

The presence of a contaminant in the environment or within human tissue alone does not mean disease will occur. Furthermore, identification of diseases for which environmental contaminants are risk factors does not mean exposure has occurred or contributed to that disease. However, extensive and collaborative data collection and research efforts across the scientific community continue to strengthen our understanding of the relationships between environmental exposures and disease. This chapter uses indicators that are tied into the environmental public health paradigm as one tool for discerning notable trends in exposure and health. First, EPA presents biomonitoring indicators to illustrate the general extent to which people are being exposed to environmental contaminants. Second, indicators of overall health status and specific diseases and conditions are used to identify potential morbidity/mortality patterns, again recognizing that environmental exposures are only one factor that could influence reported trends.

5.1.2 Establishing Linkages Between Environmental Contaminants and Health Outcomes

EPA uses the results of scientific research to help identify linkages between exposure to environmental contaminants and certain diseases, conditions, or other health outcomes. EPA relies on the possible linkages established through these types of studies to identify environmental contaminants and health outcomes of potential Agency interest (e.g., the indicators used in this chapter). Examples include radon and lung cancer; arsenic and cancer in several organs; lead and nervous system disorders; disease-causing bacteria (such as *E. coli* O157:H7) and gastrointestinal illness and death; and particulate matter and aggravation of cardiovascular and respiratory diseases. Such relationships between exposure and disease have been established through well-designed epidemiological studies with a defined or specified population (e.g., geographic location, susceptible populations, occupational exposures) and known environmental exposures.

The causes of many diseases and other health conditions are not well established. In some cases, environmental contaminants are considered important risk factors. In other cases, available data suggest that environmental exposures are important, but proof is lacking. Developing evidence that environmental contaminants cause or contribute to the incidence of adverse health effects can therefore be challenging, particularly for those effects occurring in a relatively small proportion of the population or effects with multiple causes. In cases where exposure to an environmental contaminant results in a relatively modest increase in the incidence of a disease or disorder, a large sample size for the study would be needed to detect a true relationship. In addition, there may be factors related to both the exposure and the health effect—confounding factors—that can make it difficult to detect a relationship between exposure to environmental contaminants and disease. In many cases, findings from studies in humans and/or laboratory animals may provide suggestive (rather than conclusive) evidence that exposures to environmental contaminants contribute to the incidence of a disease or disorder.

To reiterate, however, the national-scale ROE indicators do not directly link exposure with outcome and cannot be used to demonstrate causal relationships. However, when combined with other information, such as environmental monitoring data and data from toxicological, epidemiological, or clinical studies, these indicators can be an important key to better understanding the relationship between environmental contamination and health outcomes.

5.1.3 Overview of the Data

EPA draws on many resources and partnerships with other federal, state, and local agencies for the health data and statistical reports that underlie the biomonitoring and health outcome indicators used in this chapter. This chapter uses three key types of data sources, each with its own strengths and limitations:

- **Data collected from living human subjects.** This includes both questionnaire-based information (e.g., the National Center for Health Statistics' [NCHS's] National Health Interview Survey, a nationwide survey to collect data on personal and demographic characteristics, illnesses, and other topics) and biological specimens (such as NCHS's National Health and Nutrition Examination Survey, which collects and measures some chemicals in blood and urine samples). This chapter focuses on data collection activities that have a national focus and use a probability-based sampling design.

- **Vital statistics data.** Vital statistics of interest for health include births, deaths, and fetal deaths. Vital statistics data used in this chapter include NCHS's National Vital Statistics System.
- **Data from surveillance activities.** These include data from active surveillance activities such as the National Cancer Institute's Surveillance, Epidemiology, and End Results Program, which collects and publishes cancer incidence and survival data from population-based cancer registries. It also includes data from more passive collection systems, such as the Centers for Disease Control and Prevention's (CDC's) National Notifiable Disease Surveillance System, which provides information about diseases that health providers must report to state or local public health officials.

This chapter also takes advantage of several published documents that present and summarize in one place the findings from many data collection activities (e.g., NCHS's Healthy People 2010 Database). In addition, it uses some databases that provide a single point of access to a variety of reports and numeric public health data and ways to conduct analyses of those data (e.g., CDC WONDER, CDC's Wide-ranging OnLine Data for Epidemiologic Research).

The data sources used provide statistics across time, geographic areas, and/or subpopulations such as age groups, races, and ethnicities. Identifying possible differences among population subgroups, as well as evidence of whether any differences are narrowing or widening, may reveal trends needing study or intervention. This type of trend analysis is consistent with national public health goals aimed at eliminating health disparities across various groups (e.g., racial and ethnic groups, low-income populations).[2] It addresses a continuing concern that minority and/or economically disadvantaged communities frequently may be exposed disproportionately to environmental contaminants. Statistics for populations that may be particularly susceptible to environmental contaminants, such as children and pregnant women, are also examined. However, the type and level of subpopulation breakdown varies across data sets, sometimes making consistent presentation of this information difficult. Standards according to which federal agencies report race and ethnicity statistics were revised in 1997. The revised standards, which became effective in 2003, expand the race and ethnicity categories for which data are collected and are aimed at increasing comparability of data among federal data systems. As vital records used to support federal data systems continue to be revised and come into compliance with the 1997 requirements, future data reporting and comparisons will be more straightforward.

This chapter presents health statistics, including race and ethnicity subgroup categorization, as reported within the original data source documents or databases. The presentation of observed changes—temporally, spatially, or across subgroups—is descriptive, not quantitative. No statistical testing was performed (e.g., tests of statistical significance).

This chapter presents only data that meet the ROE indicator definition and criteria (see Box 1-1, p. 1-3). Note that non-scientific indicators, such as administrative and economic indicators, are not included in this definition. Thorough documentation of the indicators data sources and metadata can be found online at http://www.epa.gov/roe. All indicators were peer-reviewed during an independent peer review process (again, see http://www.epa.gov/roe for more information). Readers should not infer that the indicators included reflect the complete state of the knowledge on trends in health and exposure related to environmental exposures. Many other data sources, publications, site-specific research projects, and epidemiological studies have contributed greatly to the current understanding of health and exposure trends, but are not used because they do not meet some aspect of the ROE indicator criteria.

5.1.4 Organization of This Chapter

The rest of this chapter is organized into sections corresponding to the three questions EPA seeks to answer about trends in human health and exposure. Each section introduces the question and its importance, presents the ROE indicators selected to help answer the question, and discusses what the indicators, taken together, say about the question. The ROE indicators primarily include National Indicators, but in some cases National Indicators are broken down by EPA Region to help to answer the ROE question at a smaller geographic scale. Each section concludes by highlighting the major challenges to answering the question and identifying important information gaps.

Table 5-1 lists the indicators used to answer the three questions in this chapter and shows the locations where the indicators are presented.

[2] U.S. Department of Health and Human Services. 2000. Healthy people 2010: Understanding and improving health. Second edition. Washington, DC: U.S. Government Printing Office. <http://www.health.gov/healthypeople/>

Table 5-1. Human Exposure and Health—ROE Questions and Indicators

Question	Indicator Name	Section	Page
What are the trends in human exposure to environmental contaminants, including across population subgroups and geographic regions?	Blood Lead Level (N)	5.2.2	5-10
	Blood Mercury Level (N)	5.2.2	5-12
	Blood Cadmium Level (N)	5.2.2	5-13
	Blood Persistent Organic Pollutants Level (N)	5.2.2	5-15
	Blood Cotinine Level (N)	2.4.2	2-76
	Urinary Pesticide Level (N)	5.2.2	5-22
	Urinary Phthalate Level (N)	5.2.2	5-26
What are the trends in health status in the United States?	General Mortality (N)	5.3.2	5-33
	Life Expectancy at Birth (N)	5.3.2	5-35
	Infant Mortality (N)	5.3.2	5-36
What are the trends in human disease and conditions for which environmental contaminants may be a risk factor, including across population subgroups and geographic regions?	Cancer Incidence (N)	5.4.2	5-43
	Childhood Cancer Incidence (N)	5.4.2	5-46
	Cardiovascular Disease Prevalence (N) and Mortality (N/R)	5.4.2	5-48
	Chronic Obstructive Pulmonary Disease Prevalence (N) and Mortality (N/R)	5.4.2	5-52
	Asthma Prevalence (N)	5.4.2	5-55
	Infectious Diseases Associated with Environmental Exposures or Conditions (N)	5.4.2	5-59
	Birth Defects Prevalence and Mortality (N)	5.4.2	5-62
	Low Birthweight (N)	5.4.2	5-65
	Preterm Delivery (N)	5.4.2	5-67

N = National Indicator
N/R = National Indicator displayed at EPA Regional scale

5.2 What Are the Trends in Human Exposure to Environmental Contaminants, Including Across Population Subgroups and Geographic Regions?

5.2.1 Introduction

Understanding the extent to which human populations are being exposed to environmental contaminants helps identify those contaminants of potential public health concern and populations who may be disproportionately exposed to contaminants or uniquely vulnerable. For example, children may have disproportionately heavy exposures to environmental contaminants because they drink more water, breathe more air, and eat more food per pound or kilogram of body weight than adults; further, children may be more vulnerable to some environmental contaminants depending on the stage of development during which exposure occurs.[3,4] Evaluating exposure across certain race or ethnic groups, or other potentially susceptible subgroups, identifies possible variations in exposures. Tracking the levels of environmental contaminants in a population also enables an assessment of how exposures to those contaminants are changing in that population over time.

Referring back to the environmental public health paradigm presented in Section 5.1.1, measurements of human exposure to environmental contaminants can be made in the ambient environment (air, water, land), at the point of human contact, or after contact and contaminant entry into the human body has occurred. Box 5-1 further distinguishes the different types of exposure measures. In answering this question, the focus is on human biomonitoring, which involves the

[3] Landrigan, P.J., C.A. Kimmel, A. Correa, and B. Eskenazi. 2004. Children's health and the environment: Public health issues and challenges for risk assessment. Environ. Health Perspect. 112(2):257-265.

[4] World Health Organization. 2006. Principles for evaluating health risks in children associated with exposure to chemicals. Environmental Health Criteria 237.

measurement of human tissues or excreta for direct or indirect evidence of exposure to chemical, biological, or radiological substances. The ambient contaminant measurements presented in the media chapters are not considered here, nor can they be directly linked with biomonitoring data presented to answer this question.

Historically, human exposure has been defined as the amount of a chemical, physical, or biological contaminant at the outer boundary of the body available for exchange or intake via inhalation, ingestion, or skin or eye contact.[5] As such, human exposure to environmental contaminants has been estimated primarily through measurements of contaminant concentrations in air, water, or soil, combined with estimates of the frequency and duration of human contact with the contaminated media. These resulting exposure estimates have provided a valuable foundation for many of the regulatory and non-regulatory actions that have been taken to limit exposure to ambient contaminants. However, developments in data collection techniques and analytical methods have improved the capability to characterize human exposure via biomonitoring, which provides measurements of contaminants within the human body.

For a few environmental contaminants, particularly lead and some other metals, biomonitoring has been used for exposure characterization for a number of years. More recently, techniques for biomonitoring have been expanded to include many additional environmental contaminants. These measurements provide a tool that complements ambient measurements in characterizing human exposure to environmental contaminants. However, concentrations of environmental contaminants reported at a national level in blood, urine, or any other type of tissue cannot be used to extrapolate directly to a particular source.

The use of biological markers (or biomarkers) builds on the more traditional exposure assessment approach, providing more information on the extent to which a contaminant enters, remains, and acts in the body. Biomarker information attempts to determine the extent to which a contaminant is present in the body after entering through portals of entry such as the eyes, skin, stomach, intestines, or lungs. Given the complex set of factors that govern contaminants that are absorbed and distributed in the body, a direct measurement of the levels of a contaminant or related "marker" in the body offers more information about exposure than measured ambient levels alone.

In general, a biomarker reports the level of a substance or a marker (i.e., the product of an interaction between an agent and some target molecule or cell) present in samples collected from the body or produced by the body. *Biomarkers of exposure* measure concentrations of a contaminant, its metabolite(s), or reaction product(s) in the body fluids or tissue, most commonly blood or urine. Measurements can also be taken from a variety of other body compartments, such as feces, breast milk, hair, nails, exhaled air, and tissues obtained through biopsy or autopsy. The exposure measure used to answer this question focuses on biomarkers of exposure. Biomarkers of exposure do not predict whether biological alterations and potential health effect will result. Whether a particular exposure ultimately results in an adverse health outcome depends on a host of factors, as is described in Section 5.1.

Box 5-1. Measuring Human Exposure

Various approaches can be used to measure or estimate the levels of human exposures. No approach is best suited to all environmental contaminants, and each approach has strengths and weaknesses. Available biomonitoring data are used to answer the question on trends in human exposure to environmental contaminants.

Ambient contaminant measurements: Historically, human exposures have been estimated using environmental measurements of ambient contaminant concentrations. One limitation of ambient measurements is that the presence of a contaminant in the environment may not be fully informative regarding the extent to which individuals are exposed. In some cases, emissions data are used to model or estimate ambient concentrations.

Models of exposure: This approach combines knowledge of environmental contaminant concentrations with information on people's activities and locations (e.g., time spent working, exercising outdoors, sleeping, shopping) to account for the contact with contaminants. This approach requires knowledge of contaminant levels where people live, work, and play, as well as knowledge of their day-to-day activities. Since model output is not a direct measure of environmental conditions or exposure, it is not considered to be a true indicator of exposure.

Personal monitoring data: With personal monitoring, the monitoring device is worn by individuals as they engage in their normal day-to-day activities. This approach is most commonly used in workplace environments. Personal monitoring data provide valuable insights into the source of contaminants to which people are actually being exposed. However, a challenge with personal monitoring (as with biomonitoring) is ensuring that sufficient sampling is conducted to be representative of the population being studied. No national-scale personal monitoring data are available.

Biomonitoring data: Several environmental contaminants, notably heavy metals and some pesticides and other persistent organic pollutants, can accumulate in the body. These substances or their metabolites can be measured in human tissues or fluids such as blood or urine. These residues reflect the amount of contaminant that gets into or is present in the body, but by themselves do not provide information on how the person came into contact with the contaminant.

[5] Aldrich, T., J. Griffith, C. Cooke. 1993. Environmental epidemiology and risk assessment. New York, NY: Van Nostrand Reinhold.

5.2.2 ROE Indicators

The answer to the question on trends in human exposure relies on national-scale biomonitoring data collected as part of the Centers for Disease Control and Prevention's (CDC's) National Health and Nutrition Examination Survey (NHANES), primarily data collected from 1999 through 2002. As part of the survey, blood and urine samples are routinely collected to measure certain contaminants (or their metabolites) of public health concern. NHANES is conducted annually, but the data are combined and reported for a 2-year time period to provide more stable population estimates and to obtain adequate sample sizes for many subgroup analyses. CDC continues to process 2003–2004 and 2005–2006 survey data; raw data for the 2003–2004 survey are available for some data sets, but CDC-synthesized data and reports were not available in time for inclusion in the ROE. The chemicals in CDC's current suite of biomarkers were chosen based largely on scientific data that suggest exposure in the U.S. population, the seriousness of known or suspected health effects associated with some levels of exposure, the availability and adequacy of analytical methods, and logistical and cost considerations.[6]

Seven individual or groups of contaminants from NHANES are considered, including metals, persistent organic pollutants, pesticides, and phthalates (Table 5-2). The data presented represent data from NHANES in its entirety or a subset of the original data, with emphasis on those compounds for which CDC was able to calculate geometric means.[7] The levels of detection (LOD) presented in the indicators' exhibits vary from chemical to chemical. A chemical's LOD is the level at which the measurement has a 95 percent probability of being greater than zero. Percentile estimates that are less than the LOD for the chemical analysis are reported as "<LOD." In cases where the proportion of results below the LOD was greater than 40 percent, geometric means were not calculated and the results were reported as "NC," or not calculated.

Blood measurements for chemicals that can concentrate in lipid (e.g., dioxins, furans, PCBs, organochlorine pesticides) are presented per gram of total lipid as well as per whole weight of blood. Because these compounds are lipophilic, they concentrate in the body's lipid stores, including the lipid in blood. Blood levels reported per gram of total lipid represent the amount of these chemicals that is stored in body fat. (Blood levels per whole weight of blood are included to facilitate comparison with studies investigating exposure to these chemicals that report results in these units.) For chemicals measured in urine, levels are reported as volume in urine and per gram of creatinine. Expressing the result per gram of creatinine helps adjust for the effects of urinary dilution. For example, if one person consumed more fluids than another person, that individual's urine output is likely higher and more dilute than that of the other person.[8]

Table 5-2. ROE Indicators of Trends in Human Exposure to Environmental Contaminants

National Indicators	Section	Page
Blood Lead Level	5.2.2	5-10
Blood Mercury Level	5.2.2	5-12
Blood Cadmium Level	5.2.2	5-13
Blood Persistent Organic Pollutants Level	5.2.2	5-15
Blood Cotinine Level	2.4.2	2-76
Urinary Pesticide Level	5.2.2	5-22
Urinary Phthalate Level	5.2.2	5-26

[6] Centers for Disease Control and Prevention. 2005. Third national report on human exposure to environmental chemicals. NCEH publication no. 05-0570. <http://www.cdc.gov/exposurereport/report.htm>

[7] Geometric means are calculated by taking the log of each concentration, then calculating the mean of those log values, and finally taking the antilog of that mean. A geometric mean provides a better estimate of central tendency and is influenced less by high values than is the arithmetic mean. This type of distribution is common when measuring environmental chemicals in blood or urine. See Centers for Disease Control and Prevention. 2005. Third national report on human exposure to environmental chemicals. NCEH publication no. 05-0570. <http://www.cdc.gov/exposurereport/report.htm>

[8] Centers for Disease Control and Prevention. 2005. Third national report on human exposure to environmental chemicals. NCEH publication no. 05-0570. <http://www.cdc.gov/exposurereport/3rd/>

INDICATOR | Blood Lead Level

Lead is a naturally occurring metal found in small amounts in rock and soil. Lead has been used industrially in the production of gasoline, ceramic products, paints, metal alloys, batteries, and solder. While lead arising from the combustion of leaded gasoline was a major source of exposure in past decades, today lead-based paint and lead-contaminated dust from paint are the primary sources of lead exposure in the home. Lead levels can be measured in blood or urine.

Lead is a neurotoxic metal that affects areas of the brain that regulate behavior and nerve cell development (NRC, 1993). Its adverse effects range from subtle responses to overt toxicity, depending on how much lead is taken into the body and the age and health status of the person (CDC, 1991). Lead is one of the few pollutants for which biomonitoring and health effect data are sufficient to clearly evaluate environmental management efforts to reduce lead in the environment.

Infants, children, and fetuses are more vulnerable to the effects of lead because the blood-brain barrier is not fully developed in them (Nadakavukaren, 2000). Thus, a smaller amount of lead will have a greater effect on children than on adults. In addition, ingested lead is more readily absorbed into a child's bloodstream, while adults absorb only 10 percent. Because of lead's adverse effects on cognitive development, the Centers for Disease Control and Prevention

Exhibit 5-2. Blood lead concentrations for the U.S. population age 1 year and older by selected demographic groups, 1999-2002

			Geometric mean and selected percentiles for blood lead concentrations (µg/dL)[a]				
	Survey years	Sample size	Geometric mean	50th	75th	90th	95th
Total, age 1 year and older	1999-2000	7,970	1.7	1.6	2.4	3.8	4.9
	2001-2002	8,945	1.5	1.4	2.2	3.4	4.4
Sex							
Male	1999-2000	3,913	2.0	1.8	2.9	4.4	6.0
	2001-2002	4,339	1.8	1.7	2.7	3.9	5.3
Female	1999-2000	4,057	1.4	1.3	1.9	3.0	4.0
	2001-2002	4,606	1.2	1.1	1.8	2.6	3.6
Race and ethnicity[b]							
Black, non-Hispanic	1999-2000	1,842	1.9	1.7	2.8	4.2	5.7
	2001-2002	2,219	1.7	1.6	2.5	4.2	5.7
Mexican American	1999-2000	2,742	1.8	1.8	2.7	4.2	5.8
	2001-2002	2,268	1.5	1.5	2.2	3.6	5.4
White, non-Hispanic	1999-2000	2,716	1.6	1.6	2.4	3.6	5.0
	2001-2002	3,806	1.4	1.4	2.1	3.1	4.1
Age group							
1-5 years	1999-2000	723	2.2	2.2	3.3	4.8	7.0
	2001-2002	898	1.7	1.5	2.5	4.1	5.8
6-11 years	1999-2000	905	1.5	1.3	2.0	3.3	4.5
	2001-2002	1,044	1.3	1.1	1.6	2.7	3.7
12-19 years	1999-2000	2,135	1.1	1.0	1.4	2.3	2.8
	2001-2002	2,231	0.9	0.8	1.2	1.9	2.7
20+ years	1999-2000	4,207	1.8	1.7	2.5	3.9	5.2
	2001-2002	4,772	1.6	1.6	2.2	3.6	4.6

[a]Refer to CDC 2005 for confidence intervals for reported values.

[b]Other racial and ethnic groups are included in the "total" only.

Data source: *CDC, 2005*

(CDC) have defined an elevated blood lead level as equal to or greater than 10 micrograms per deciliter (µg/dL) for children under 6 years of age (CDC, 2005).

This indicator is based on data collected by the National Health and Nutrition Examination Survey (NHANES). NHANES is a series of surveys conducted by CDC's National Center for Health Statistics that is designed to collect data on the health and nutritional status of the civilian, non-institutionalized U.S. population using a complex, stratified, multistage, probability-cluster design. CDC began monitoring blood lead in 1976 as part of NHANES II, which covered the period from 1976 through 1980. Blood lead was also monitored in NHANES III, which covered the period between 1988 and 1994. CDC's National Center for Environmental Health conducted the laboratory analyses for the biomonitoring samples. Beginning in 1999, NHANES became a continuous and annual national survey, visiting 15 U.S. locations per year and surveying and reporting for approximately 5,000 people annually.

What the Data Show

The overall geometric mean blood lead levels among all participants age 1 year and older from NHANES 1999-2000 and 2001-2002 were 1.7 µg/dL and 1.5 µg/dL, respectively (Exhibit 5-2). Adults 20 years and older had a geometric mean lead level of 1.6 µg/dL during the 2001-2002 NHANES. For this same period, males and females had geometric mean lead levels of 1.8 µg/dL and 1.2 µg/dL, respectively. For non-Hispanic blacks, Mexican Americans, and non-Hispanic whites during 2001-2002, the geometric mean lead levels were 1.7, 1.5, and 1.4 µg/dL, respectively. The geometric mean blood levels among every age, race, and ethnic group, as well as for both males and females, declined in the most recent 2001-2002 survey. Of all age groups, children age 1 to 5 had the highest geometric mean lead level, at 1.7 µg/dL. However, this age group also showed the largest decline between 1999-2000 and 2001-2002 (2.2 µg/dL to 1.7 µg/dL). Children age 6 to 11 and 12 to 19 had reported geometric mean lead levels of 1.3 and 0.9 µg/dL, respectively, for the 2001-2002 survey.

Blood lead levels have declined steadily since NHANES surveillance of blood lead levels across the U.S. began in 1976. NHANES II (1976-1980) reported a geometric mean blood lead level of 14.9 µg/dL among children age 1 to 5, the population at the highest risk for lead exposure and effects; just over 88 percent of this high-risk population had blood lead levels greater than or equal to 10 µg/dL (CDC, 2004a). Data collected from 1991 to 1994 as part of NHANES III (phase 2) showed that the geometric mean blood lead level for children age 1 to 5 was 2.7 µg/dL, with 4.4 percent of children age 1 to 5 having blood lead levels greater than or equal to 10 µg/dL (CDC, 2005). Children age 1 to 5 whose blood was sampled as part of the 1999-2002 survey had a geometric mean blood lead level of 1.9 µg/dL, with 1.6 percent of the children having blood lead levels greater than or equal to 10 µg/dL (CDC, 2005). (Data not shown.)

Indicator Limitations

- Because the data from NHANES 1999-2000 and 2001-2002 represent only two survey periods, changes in estimates between the two time periods do not necessarily reflect a trend. Earlier data sets are available (e.g., NHANES III), but the data are not directly comparable to NHANES 1999-2002. As CDC releases additional survey results (e.g., 2003-2004), it will become possible to more fully evaluate trends (CDC, 2002, 2004b).

Data Source

Data used for this indicator were extracted from two CDC reports that present results of the ongoing NHANES (CDC, 2004a, 2005). The underlying laboratory data supporting CDC's reports are available online in SAS® transport file format at http://www.cdc.gov/nchs/about/major/nhanes/datalink.htm.

References

CDC (Centers for Disease Control and Prevention). 2005. Third national report on human exposure to environmental chemicals. NCEH publication no. 05-0570. <http://www.cdc.gov/exposurereport/report.htm>

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CDC. 2004b. NHANES analytic guidelines. June 2004 version. <http://www.cdc.gov/nchs/data/nhanes/nhanes_general_guidelines_june_04.pdf>

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NRC (National Research Council). 1993. Measuring lead exposure in infants, children, and other sensitive populations. Washington, DC: National Academies Press.

INDICATOR | Blood Mercury Level

Mercury is a naturally occurring metal. However, through many industrial processes (e.g., chemical manufacturing operations, coal combustion), mercury is widespread and persistent in the environment. It is found in elemental form and in various organic compounds and complexes. Methylmercury (an organic form) can accumulate in the food chain in aquatic systems and lead to high concentrations in predatory fish. Consumption of contaminated fish is the major source of human exposure to methylmercury in the U.S. (NRC, 2000).

The human health effects of mercury are diverse and depend on the forms of mercury encountered and the severity and length of exposure. Fetuses and children may be more susceptible to mercury than adults, with concern for the occurrence of developmental and neurological health effects (NRC, 2000). Prenatal exposures interfere with the growth and migration of neurons and have the potential to cause irreversible damage to the developing central nervous system.

This indicator quantifies the blood mercury levels (includes organic and inorganic) among U.S. women age 16 to 49 and children age 1 to 5, using data from the 1999-2002 National Health and Nutrition Examination Survey (NHANES). NHANES does not report blood mercury data for adult males. NHANES is a series of surveys conducted by the Centers for Disease Control and Prevention's (CDC's) National Center for Health Statistics that is designed to collect data on the health and nutritional status of the civilian, non-institutionalized U.S. population using a complex, stratified, multistage, probability-cluster design. CDC's National Center for Environmental Health conducted the laboratory analyses for the biomonitoring

Exhibit 5-3. Blood mercury concentrations for U.S. women age 16-49 years and children (male and female) age 1-5 years by selected demographic groups, 1999-2002

			Geometric mean and selected percentiles for mercury concentrations (µg/L)[a]				
	Survey years	Sample size	Geometric mean	50th	75th	90th	95th
Women age 16-49 years							
Total, women age 16-49 years	1999-2000	1,709	1.0	0.9	2.0	4.9	7.1
	2001-2002	1,928	0.8	0.7	1.7	3.0	4.6
Race and ethnicity							
Black, non-Hispanic	1999-2000	370	1.4	1.3	2.6	4.8	5.9
	2001-2002	436	1.1	1.1	1.8	3.2	4.1
Mexican American	1999-2000	579	0.8	0.9	1.4	2.6	4.0
	2001-2002	527	0.7	0.7	1.1	2.1	3.5
White, non-Hispanic	1999-2000	588	0.9	0.9	1.9	5.0	6.9
	2001-2002	806	0.8	0.8	1.5	3.0	4.6
Children age 1-5 years							
Total, children age 1-5 years	1999-2000	705	0.3	0.3	0.5	1.4	2.3
	2001-2002	872	0.3	0.3	0.7	1.2	1.9
Sex							
Male	1999-2000	387	0.3	0.2	0.5	1.1	2.1
	2001-2002	440	0.3	0.3	0.6	1.3	1.7
Female	1999-2000	318	0.4	0.2	0.8	1.6	2.7
	2001-2002	432	0.3	0.3	0.7	1.3	2.6
Race and ethnicity							
Black, non-Hispanic	1999-2002	424	0.5	0.5	0.9	1.5	2.4
Mexican American	1999-2002	526	0.4	0.3	0.6	1.4	1.9
White, non-Hispanic	1999-2002	447	0.3	0.2	0.5	1.2	1.8

[a]Refer to CDC, 2005, for confidence intervals for reported values.

Data source: *CDC, 2004a, 2005*

INDICATOR | Blood Mercury Level *(continued)*

samples. Beginning in 1999, NHANES became a continuous and annual national survey. Data for 1999–2000 and 2001–2002 are presented here as a baseline, with the intent of reporting trends across time as more data become available in the future.

What the Data Show

Exhibit 5-3 presents the geometric mean and four percentiles of blood mercury for selected populations sampled during NHANES 1999–2000 and 2001–2002. For women age 16–49 years there was a small decline in geometric mean blood mercury levels from 1999–2000 and 2001–2002 (1.0 and 0.8 micrograms per liter [µg/L], respectively). Decreases occurred for each of the four percentiles, but were most pronounced at the 90th and especially 95th percentiles. Of women tested between 1999 and 2002, 5.7 percent had mercury levels measured between 5.8 and 58 µg/L (data not shown). For children age 1 to 5, the geometric mean remained the same at 0.3 µg/L.

When the geometric means are stratified across three racial/ethnic groups, black, non-Hispanic women age 16 to 49 had the highest levels during both the 1999–2000 and 2001–2002 surveys (1.4 and 1.1 µg/L, respectively), followed by white non-Hispanics (0.9 and 0.8 µg/L, respectively), and Mexican Americans (0.8 and 0.7 µg/L, respectively). Among children age 1 to 5, black non-Hispanics have the highest geometric mean between 1999 and 2002 (0.5 µg/L), followed by Mexican Americans (0.4 µg/L) and white non-Hispanics (0.3 µg/L) (CDC, 2004a).

Indicator Limitations

- Because the data from NHANES 1999–2000 and 2001–2002 represent only two survey periods, changes in estimates between the two time periods do not necessarily reflect a trend. As CDC releases additional survey results (e.g., 2003–2004) it will become possible to more fully evaluate trends (CDC, 2002, 2004b).
- Generally recognized guidelines for blood levels of mercury have not been established.

Data Sources

Data used for this indicator were extracted from two CDC reports that present results of the ongoing NHANES (CDC, 2004a, 2005). The underlying laboratory data supporting CDC's reports are available online in SAS® transport file format at http://www.cdc.gov/nchs/about/major/nhanes/datalink.htm.

References

CDC (Centers for Disease Control and Prevention). 2005. Third national report on human exposure to environmental chemicals. NCEH publication no. 05-0570. <http://www.cdc.gov/exposurereport/report.htm>

CDC. 2004a. Blood mercury levels in young children and childbearing-aged women—United States, 1999–2002. MMWR 53:1018–1020. <http://www.cdc.gov/mmwr/PDF/wk/mm5343.pdf>

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CDC. 2002. NHANES 1999–2000 addendum to the NHANES III analytic guidelines. Updated August 30, 2002. <http://www.cdc.gov/nchs/data/nhanes/guidelines1.pdf>

NRC (National Research Council). 2000. Toxicological effects of methylmercury. Washington, DC: National Academies Press.

INDICATOR | Blood Cadmium Level

Cadmium is a metal that is usually found in nature combined with oxygen, chlorine, or sulfur. Cadmium enters the environment from the weathering of rocks and minerals that contain cadmium. Exposure to cadmium can occur in occupations such as mining or electroplating, where cadmium is produced or used. Cadmium exposure can also occur from exposure to cigarette smoke (CDC, 2005).

Cadmium and its compounds are toxic to humans and animals. Once absorbed into the human body, cadmium can accumulate in the kidneys and remain in the body for decades. Chronic exposure to cadmium can result in serious kidney damage. Osteomalacia, a bone disorder similar to rickets, is also associated with long-term ingestion of cadmium. Acute airborne exposure, as occurs from welding on cadmium-alloy metals, can result in swelling (edema) and scarring (fibrosis) of the lungs (CDC, 2005).

This indicator reflects blood cadmium concentrations in micrograms per liter (µg/L) for the U.S. population, age 1 year and older, as measured in the 1999–2002 National Health and Nutrition Examination Survey (NHANES). NHANES is a series of surveys conducted by the Centers for Disease Control and Prevention's (CDC's) National Center for Health Statistics that is designed to collect data on the health and nutritional status of the civilian, noninstitutionalized U.S. population using a complex, stratified, multistage, probability-cluster design. CDC's National Center for Environmental Health conducted the laboratory analyses for the biomonitoring samples. Beginning in 1999,

INDICATOR | Blood Cadmium Level *(continued)*

NHANES became a continuous and annual national survey; biomonitoring for certain environmental chemicals also was implemented. Data for 1999–2000 and 2001–2002 are presented here as a baseline, with the intent of reporting trends across time as more data become available in the future.

What the Data Show

Exhibit 5-4 presents the geometric means and selected percentiles for blood cadmium among participants age 1 year and older from NHANES 1999–2000 and 2001–2002. During the 2001–2002 survey, the overall geometric mean blood cadmium level was not calculated because of the high number of samples that were below the method's limit of detection. However, the blood cadmium levels at the four different percentiles (50th, 75th, 90th, and 95th) are very similar across the two survey periods, with levels ranging between 0.3 and 1.4 µg/L. The blood cadmium measurements were similar among males and females, as well as among the racial or ethnic groups sampled across both time periods.

During the 1999–2000 survey, the overall geometric mean among participants age 20 or older was slightly higher (0.5 µg/L) than the geometric mean among the 12–19 age group (0.3 µg/L). Compared to participants in the other age groups, those older than 20 years had higher cadmium levels for each of the four selected percentiles

Exhibit 5-4. Blood cadmium concentrations for the U.S. population age 1 year and older by selected demographic groups, 1999-2002

			Geometric mean and selected percentiles for cadmium concentrations (µg/L)[a, b, c]				
	Survey years	Sample size	Geometric mean	50th	75th	90th	95th
Total, age 1 year and older	1999-2000	7,970	0.4	0.3	0.6	1.0	1.3
	2001-2002	8,945	NC	0.3	0.4	0.9	1.3
Sex							
Male	1999-2000	3,913	0.4	0.4	0.6	1.0	1.3
	2001-2002	4,339	NC	0.3	0.4	0.9	1.4
Female	1999-2000	4,057	0.4	0.3	0.6	1.0	1.3
	2001-2002	4,606	NC	0.3	0.5	1.0	1.4
Race and ethnicity[d]							
Black, non-Hispanic	1999-2000	1,842	0.4	0.3	0.6	1.0	1.4
	2001-2002	2,219	NC	<LOD	0.4	1.0	1.4
Mexican American	1999-2000	2,742	0.4	0.4	0.4	0.7	1.1
	2001-2002	2,268	NC	<LOD	0.3	0.6	1.0
White, non-Hispanic	1999-2000	2,716	0.4	0.4	0.5	1.0	1.3
	2001-2002	3,806	NC	<LOD	0.5	0.9	1.4
Age group							
1-5 years	1999-2000	723	NC	<LOD	0.3	0.4	0.4
	2001-2002	898	NC	<LOD	<LOD	<LOD	0.3
6-11 years	1999-2000	905	NC	<LOD	0.3	0.4	0.4
	2001-2002	1,044	NC	<LOD	<LOD	<LOD	0.4
12-19 years	1999-2000	2,135	0.3	0.3	0.3	0.8	1.1
	2001-2002	2,231	NC	<LOD	0.3	0.4	0.8
20+ years	1999-2000	4,207	0.5	0.4	0.6	1.0	1.5
	2001-2002	4,772	NC	0.3	0.6	1.1	1.6

[a]NC = not calculated; the proportion of results below the limit of detection was too high to provide a valid result.

[b]LOD = below the limit of detection (LOD) of the analytical method (cadmium LOD = 0.04 µg/L).

[c]Refer to CDC, 2005, for confidence intervals for reported values.

[d]Other racial and ethnic groups are included in the "total" only.

Data source: *CDC, 2005*

INDICATOR | Blood Cadmium Level *(continued)*

during both survey periods. During the 1999-2000 survey, approximately half of all participants under the age of 12 had non-detectable blood cadmium concentrations. This proportion increased to about 90 percent during the 2001-2002 survey.

Indicator Limitations

- Because the data from NHANES 1999-2000 and 2001-2002 represent only two survey periods, changes in estimates between the two time periods do not necessarily reflect a trend. As CDC releases additional survey results (e.g., 2003-2004), it will become possible to more fully evaluate trends (CDC, 2002, 2004).
- Generally recognized guidelines for blood levels of cadmium have not been established.

Data Sources

Data used for this indicator were extracted from the CDC report that presents results of the ongoing NHANES (CDC, 2005). The underlying laboratory data supporting CDC's report are available online in SAS® transport file format at http://www.cdc.gov/nchs/about/major/nhanes/datalink.htm.

References

CDC (Centers for Disease Control and Prevention). 2005. Third national report on human exposure to environmental chemicals. NCEH publication no. 05-0570. <http://www.cdc.gov/exposurereport/report.htm>

CDC. 2004. NHANES analytic guidelines. June 2004 version. <http://www.cdc.gov/nchs/data/nhanes/nhanes_general_guidelines_june_04.pdf>

CDC. 2002. NHANES 1999-2000 addendum to the NHANES III analytic guidelines. Updated August 30, 2002. <http://www.cdc.gov/nchs/data/nhanes/guidelines1.pdf>

INDICATOR | Blood Persistent Organic Pollutants Level

Persistent organic pollutants (POPs) are manmade organic chemicals that remain in the environment for years or decades. POPs are of special concern because they often remain toxic for decades or longer after release to the environment. The more persistent a toxic chemical is, the greater the probability for human exposure over time. Because they circulate globally long after being released into the environment, POPs are often detected in locations far from the original source (U.S. EPA, 2004a).

One of the major sources of POPs exposure among the general population is food. Food contamination begins with contaminated soil and/or plants, but is of greatest concern to humans as the POPs move up the food chain into animals. Because POPs typically accumulate in fatty tissue and are slow to be metabolized, they bioconcentrate (i.e., increase in concentration) with each trophic level. Therefore, foods such as dairy products, eggs, animal fats, and some types of fish are more likely to contain greater concentrations of POPs than fruits, vegetables, and grains. POPs have been linked to adverse health effects such as cancer, nervous system damage, reproductive disorders, and disruption of the immune system in both humans and animals (U.S. EPA, 2004a).

This indicator presents data from the Centers for Disease Control and Prevention's (CDC's) National Health and Nutrition Examination Survey (NHANES) 1999-2000 and 2001-2002. NHANES is a series of surveys conducted by CDC's National Center for Health Statistics that is designed to collect data on the health and nutritional status of the civilian, non-institutionalized U.S. population using a complex, stratified, multistage, probability-cluster design. CDC's National Center for Environmental Health conducted the laboratory analyses for the biomonitoring samples. Beginning in 1999, NHANES became a continuous and annual national survey; biomonitoring for certain environmental chemicals also was implemented. These data are presented here as a baseline, with the intent of reporting trends over larger time periods in the future. Blood levels of POPs or their metabolites were measured in NHANES participants age 12 or older. This indicator includes the following three broad classes of POPs:

- Organochlorine pesticides
- Polychlorinated dibenzo-p-dioxins (dioxins) and polychlorinated dibenzo-p-furans (furans)
- Polychlorinated biphenyls (PCBs)

Organochlorine pesticides were first introduced in the 1940s. Because of their environmental persistence, EPA banned most uses of these chemicals during the 1970s and 1980s. However, many other countries still produce and/or use organochlorines. These fat-soluble chemicals are most commonly absorbed through fatty foods. These pesticides are associated with effects to the central nervous system at acute exposure levels and potential carcinogenic effects with long-term exposure (Reigart and Roberts, 1999). This indicator includes eight organochlorine pesticides that

Exhibit 5-5. Blood concentrations of selected organochlorine pesticides and metabolites for the U.S. population age 12 years and older, lipid-adjusted and whole weight, 1999-2002

			Geometric mean and selected percentiles for organochlorine pesticide metabolite concentrations (ng/g)[a,b,c]				
	Survey years	Sample size	Geometric mean	50th	75th	90th	95th
Aldrin							
Lipid-adjusted	2001-2002	2,275	NC	<LOD	<LOD	<LOD	<LOD
Whole weight	2001-2002	2,275	NC	<LOD	<LOD	<LOD	<LOD
Chlordane							
Oxychlordane							
Lipid-adjusted	1999-2000	1,661	NC	<LOD	20.6	34.4	44.8
	2001-2002	2,249	11.4	11.1	21.7	36.3	49.7
Whole weight	1999-2000	1,661	NC	<LOD	0.13	0.26	0.31
	2001-2002	2,249	0.07	0.07	0.14	0.25	0.35
trans-Nonachlor							
Lipid-adjusted	1999-2000	1,933	18.3	17.8	31.9	55.1	79.4
	2001-2002	2,286	17.0	17.9	33.7	56.3	78.2
Whole weight	1999-2000	1,933	0.11	0.11	0.21	0.37	0.54
	2001-2002	2,286	0.10	0.11	0.22	0.39	0.59
DDT/DDE							
p,p'-DDE							
Lipid-adjusted	1999-2000	1,964	260	226	537	1,150	1,780
	2001-2002	2,298	295	250	597	1,400	2,320
Whole weight	1999-2000	1,964	1.54	1.31	3.49	7.49	11.6
	2001-2002	2,298	1.81	1.57	3.97	8.81	15.4
p,p'-DDT							
Lipid-adjusted	1999-2000	1,679	NC	<LOD	<LOD	<LOD	28.0
	2001-2002	2,305	NC	<LOD	<LOD	<LOD	26.5
Whole weight	1999-2000	1,679	NC	<LOD	<LOD	<LOD	0.17
	2001-2002	2,305	NC	<LOD	<LOD	<LOD	0.18
o,p'-DDT							
Lipid-adjusted	1999-2000	1,669	NC	<LOD	<LOD	<LOD	<LOD
	2001-2002	2,279	NC	<LOD	<LOD	<LOD	<LOD
Whole weight	1999-2000	1,669	NC	<LOD	<LOD	<LOD	<LOD
	2001-2002	2,279	NC	<LOD	<LOD	<LOD	<LOD
Dieldrin							
Lipid-adjusted	2001-2002	2,159	NC	<LOD	<LOD	15.2	20.3
Whole weight	2001-2002	2,159	NC	<LOD	<LOD	0.11	0.15
Endrin							
Lipid-adjusted	2001-2002	2,187	NC	<LOD	<LOD	<LOD	5.1
Whole weight	2001-2002	2,187	NC	<LOD	<LOD	<LOD	0.02

See notes at end of table.

Continued

Exhibit 5-5 (continued)**.** Blood concentrations of selected organochlorine pesticides and metabolites for the U.S. population age 12 years and older, lipid-adjusted and whole weight, 1999-2002

			Geometric mean and selected percentiles for organochlorine pesticide metabolite concentrations (in ng/g)[a,b,c]				
	Survey years	Sample size	Geometric mean	50th	75th	90th	95th
Heptachlor							
Heptachlor epoxide							
Lipid-adjusted	1999-2000	1,589	NC	<LOD	<LOD	15.3	23.9
	2001-2002	2,259	NC	<LOD	<LOD	14.8	21.6
Whole weight	1999-2000	1,589	NC	<LOD	<LOD	0.11	0.18
	2001-2002	2,259	NC	<LOD	<LOD	0.10	0.15
Hexachlorobenzene (HCB)							
Lipid-adjusted	1999-2000	1,702	NC	<LOD	<LOD	<LOD	<LOD
	2001-2002	2,277	NC	<LOD	<LOD	<LOD	<LOD
Whole weight	1999-2000	1,702	NC	<LOD	<LOD	<LOD	<LOD
	2001-2002	2,277	NC	<LOD	<LOD	<LOD	<LOD
Mirex							
Lipid-adjusted	1999-2000	1,853	NC	<LOD	<LOD	<LOD	<LOD
	2001-2002	2,257	NC	<LOD	<LOD	15.8	57.1
Whole weight	1999-2000	1,853	NC	<LOD	<LOD	<LOD	<LOD
	2001-2002	2,257	NC	<LOD	<LOD	0.10	0.41

[a]NC = not calculated; the proportion of results below the limit of detection was too high to provide a valid result.

[b]<LOD = below the limit of detection (LOD) of the analytical method (see CDC, 2005, for chemical-specific LODs).

[c]Refer to CDC, 2005, for confidence intervals for reported values.

Data source: *CDC, 2005*

were measured in NHANES 1999-2000 and 2001-2002; data for three of these pesticides (aldrin, dieldrin, and endrin) first became available with the release of results from NHANES 2001-2002 (CDC, 2005).

- **Aldrin and dieldrin.** These two pesticides were widely used from the 1950s until 1970, when EPA prohibited most agricultural uses. However, they continued to be used to control termites until that use was prohibited in 1987. Aldrin rapidly converts to dieldrin in the environment or after being ingested or absorbed into the body. Dieldrin is more persistent and often accumulates in fatty tissues (CDC, 2005).
- **Chlordane and heptachlor.** EPA banned these pesticides in 1988. Within the body, chlordane is metabolized to oxychlordane and *trans*-nonachlor, and heptachlor is metabolized to heptachlor epoxide (CDC, 2003). Chlordane was commonly used against termites and on some agricultural crops and heptachlor was used primarily against soil insects and termites (Ritter et al., n.d.).
- **DDT.** Dichlorodiphenyltrichlorethane, or DDT, was banned in the U.S. in 1973 but is still produced in other countries, where it is used primarily to control mosquitoes. In the body or the environment, DDT breaks down to DDE (dichlorodiphenyldichloroethane), a more persistent chemical. DDT or DDE in the human body may reflect either a relatively recent exposure or cumulative past exposures (CDC, 2005).
- **Endrin.** Endrin is a stereoisomer (i.e., a molecule that is a mirror image of another molecule with the same molecular formula) of dieldrin. Endrin production was discontinued in 1986, primarily because of its persistence in the environment. Unlike many other organochlorine pesticides, endrin does not readily accumulate in body tissues and is metabolized and eliminated from the body relatively quickly (CDC, 2005).
- **Hexachlorobenzene** (HCB) was commonly used as a pesticide until 1965. HCB was also used in the past as a fungicide to protect wheat seeds, and for a variety of industrial purposes, including rubber, aluminum,

Exhibit 5-6. Blood concentrations of selected polychlorinated dibenzo-p-dioxins (dioxins), polychlorinated dibenzofurans (furans), and dioxin-like polychlorinated biphenyls (PCBs) for the U.S. population age 20 years and older, lipid-adjusted and whole weight, 1999-2002[a,b]

			Geometric mean and selected percentiles for dioxin, furan, and PCB concentrations[c,d,e]				
	Survey years	Sample size	Geometric mean	50th	75th	90th	95th
Dioxins (pg/g)							
1,2,3,4,6,7,8,9-OCDD							
Lipid-adjusted	1999-2000	1,254	NC	<LOD	445	704	948
	2001-2002	1,171	346	333	571	939	1,260
Whole weight	1999-2000	1,254	NC	<LOD	2.80	4.57	6.20
	2001-2002	1,171	2.23	2.17	3.86	6.46	9.11
1,2,3,4,6,7,8-HpCDD							
Lipid-adjusted	1999-2000	1,237	NC	<LOD	61.9	92	119
	2001-2002	1,220	39	40.2	68.7	115	147
Whole weight	1999-2000	1,237	NC	<LOD	0.39	0.61	0.80
	2001-2002	1,220	0.25	0.27	0.44	0.78	1.03
1,2,3,6,7,8-HxCDD							
Lipid-adjusted	1999-2000	1,237	NC	<LOD	36.1	62.8	75.6
	2001-2002	1,234	34.6	39.2	60.7	95.2	127
Whole weight	1999-2000	1,237	NC	<LOD	0.23	0.40	0.52
	2001-2002	1,234	0.22	0.25	0.41	0.66	0.87
Furans (pg/g)							
1,2,3,4,6,7,8-HpCDF							
Lipid-adjusted	1999-2000	1,109	NC	<LOD	<LOD	14.2	18.4
	2001-2002	1,219	9.6	10.3	14.5	21.3	27.1
Whole weight	1999-2000	1,109	NC	<LOD	<LOD	0.09	0.11
	2001-2002	1,219	0.06	0.06	0.09	0.13	0.18
PCBs (units vary)							
PCB 126 (pg/g)							
Lipid-adjusted	1999-2000	1,238	NC	<LOD	30.8	57.1	89.5
	2001-2002	1,226	22.7	24.5	40.8	69.3	108
Whole weight	1999-2000	1,238	NC	<LOD	0.20	0.38	0.59
	2001-2002	1,226	0.15	0.16	0.27	0.48	0.73
PCB 169 (pg/g)							
Lipid-adjusted	1999-2000	1,240	NC	<LOD	<LOD	36.4	47.8
	2001-2002	1,223	17.9	19	33.1	50.0	60.7
Whole weight	1999-2000	1,240	NC	<LOD	<LOD	0.24	0.30
	2001-2002	1,223	0.12	0.13	0.22	0.34	0.42

See notes at end of table.

Continued

Exhibit 5-6 (continued)**.** Blood concentrations of selected polychlorinated dibenzo-p-dioxins (dioxins), polychlorinated dibenzofurans (furans), and dioxin-like polychlorinated biphenyls (PCBs) for the U.S. population age 20 years and older, lipid-adjusted and whole weight, 1999-2002[a,b]

			Geometric mean and selected percentiles for dioxin, furan, and PCB concentrations[c,d,e]				
	Survey years	Sample size	Geometric mean	50th	75th	90th	95th
PCBs (units vary)							
PCB 138 & 158 (ng/g)							
Lipid-adjusted	1999-2000	1,261	NC	<LOD	<LOD	54.7	72.8
	2001-2002	1,545	23.3	23.9	44.6	73.8	99.5
Whole weight	1999-2000	1,261	NC	<LOD	<LOD	0.36	0.49
	2001-2002	1,545	0.15	0.15	0.29	0.51	0.68
PCB 153 (ng/g)							
Lipid-adjusted	1999-2000	1,258	NC	<LOD	<LOD	83.2	122
	2001-2002	1,549	32.6	35	62.8	99.5	132
Whole weight	1999-2000	1,258	NC	<LOD	<LOD	0.56	0.79
	2001-2002	1,549	0.21	0.22	0.41	0.67	0.90
PCB 180 (ng/g)							
Lipid-adjusted	1999-2000	1,257	NC	<LOD	41	65.5	83.8
	2001-2002	1,547	23	26.4	46.7	74	90.7
Whole weight	1999-2000	1,257	NC	<LOD	0.27	0.44	0.56
	2001-2002	1,547	0.15	0.17	0.30	0.49	0.64

[a]The 1999-2000 subsample included those aged 12-19 years and aged 20 years and older. The 2001-2002 subsample does not include the 12-19 year-old age group. To enable comparisons, this table presents results for the 20 and older age group only.

[b]This table only includes individual congeners detected with sufficient frequency to calculate a geometric mean.

[c]<LOD = below the limit of detection (LOD) of the analytical method (see CDC, 2005, for chemical-specific LODs).

[d]NC = not calculated; the proportion of results below the limit of detection was too high to provide a valid result.

[e]Refer to CDC, 2005, for confidence intervals for reported values.

Data source: *CDC, 2005*

and dye production and wood preservation (U.S. EPA, 2004b). EPA canceled registered use in 1984; however, HCB is still formed as a byproduct during manufacturing of other chemicals and pesticides (U.S. EPA, 2004b).

- **Mirex** has not been produced or used in the U.S. since 1978. It was used primarily in the southern U.S. to control fire ants. The primary source of exposure is dietary, most often through consumption of fish (U.S. EPA, 2004c).

Dioxins and furans are similar classes of chlorinated aromatic chemicals, usually generated as pollutants or byproducts. In the environment, dioxins and furans occur as a mixture of about 20 compounds (termed "congeners"). The half-lives of these congeners range from roughly 3 to 19 years (CDC, 2005). Human exposure occurs primarily through food; other sources of exposure include industrial accidents, burning of PCBs contaminated with dioxins and furans, burning of many plastics such as PVC, and spraying or unintended releases of contaminated herbicides such as Agent Orange. The detection of dioxins and furans in human blood can reflect either recent or past exposures (CDC, 2005).

Researchers continue to study the potential adverse health effects associated with dioxins and furans. Studies of individual congeners have shown immunotoxic, developmental/reproductive, and other systemic effects. The effects of individual congeners in humans are difficult to determine, since exposures are more likely be to mixtures of several congeners. The dioxin congener TCDD (2,3,7,8-tetrachlorodibenzo-p-dioxin) is the most toxic form of dioxin and is classified as a known human carcinogen (IARC, 1997). Uncertainties remain, however, about the levels and mechanisms involved in producing harmful effects in humans.

INDICATOR | Blood Persistent Organic Pollutants Level *(continued)*

PCBs are chlorinated aromatic hydrocarbons used in a variety of industries as electrical insulating and heat exchange fluids. PCBs are composed of mixtures of up to 209 different chlorinated congeners. U.S. production of PCBs peaked in the early 1970s; PCBs were banned in 1979. Sources of exposure for the general population include releases from waste sites and fires involving transformers, ingestion of foods contaminated by PCBs, and migration from packaging materials. PCBs typically accumulate in fatty tissues (ATSDR, 2000).

The detection of PCBs in human blood can reflect either recent or past exposures. PCBs with higher degrees of chlorination persist in the human body from several months to years after exposure. Coplanar and mono-ortho substituted PCBs exhibit health effects similar to dioxins. The human health effects of PCBs include changes in liver function, elevated lipids, and gastrointestinal cancers (CDC, 2005).

What the Data Show

Organochlorine Pesticides

Exhibit 5-5 presents the lipid-adjusted and whole weight geometric means and four percentile values for selected organochlorine pesticide metabolites measured in blood. The overall geometric mean for *p,p'*-DDE (a metabolite for DDT) during the 1999-2000 survey was 260 nanograms per gram (ng/g), compared to 295 ng/g in 2001-2002. During the most recent survey (2001-2002), the geometric mean for *trans*-nonachlor (a component of technical-grade chlordane) was 17 ng/g, compared with 18.3 ng/g in 1999-2000. Aldrin, dieldrin, endrin, heptachlor epoxide (the metabolite for heptachlor), HCB, and mirex were not measured with sufficient frequency above the limit of detection to calculate a geometric mean.

Geometric mean blood concentrations of *p,p'*-DDE were compared among demographic groups after adjustment for the covariates of race/ethnicity, age, and gender. For samples collected between 1999 and 2002, the 12-19 year age group had less than half the blood *p,p'*-DDE level compared to the 20 years or older age group (CDC, 2005). The lipid-adjusted geometric mean level in Mexican Americans was 652 ng/g during the most recent survey, more than two and one-half times higher than levels in non-Hispanic whites and two times higher than levels in non-Hispanic blacks. It is unknown whether differences in geometric mean blood *p,p'*-DDE concentrations between different age groups or racial/ethnic groups represent differences in exposure, body size relationships, or metabolism (CDC, 2005) (data not shown).

Dioxins and Furans

In the U.S., quantifiable emissions of dioxin-like compounds from all known sources have decreased by an estimated 90 percent between 1987 and 2000 (U.S. EPA, 2006). Values reported in NHANES 1999-2000 and 2001-2002 support that estimated decline (CDC, 2005). For example, among the entire NHANES 1999-2000 sample population, TCDD (generally considered the most toxic dioxin) was detected less than 1 percent of the time (CDC, 2003). During 2001-2002, only a small number of the dioxin and furan congeners analyzed were detected frequently enough for geometric means to be calculated (Exhibit 5-6). TCDD continued to be among the list of congeners analyzed in NHANES 2001-2002, though only the 95th percentiles for women and non-Hispanic blacks could be characterized: 6.4 and 7.4 picograms per gram (pg/g) TCDD lipid-adjusted, respectively (data not shown). From NHANES 1999-2000, none of the six dioxin or nine furan congeners measured in the blood were detected with sufficient frequency to calculate a geometric mean.

In general, the more highly chlorinated dioxin and furan congeners were the main contributors to the human body burden. The higher concentrations of these congeners in human samples are a result of their greater persistence in the environment, bioaccumulation in the food chain, resistance to metabolic degradation, and greater solubility in body fat (CDC, 2005).

PCBs

During the NHANES 1999-2000 subsample period, none of the three coplanar and 25 other PCB congeners were measured in blood with sufficient frequency above the limit of detection to calculate a geometric mean. The frequency of detection of the eight mono-ortho substituted PCBs ranged from 2 to 47 percent (CDC, 2003). Coplanar PCB congeners 169 and 126, which exhibit dioxin-like toxicity, had a detection rate above 5 percent (CDC, 2003). In the 2001-2002 survey, a total of 12 dioxin-like PCB compounds, three coplanar PCBs and nine mono-ortho-substituted PCBs, were measured in blood. A total of 25 non-dioxin-like PCBs were also included in the 2001-2002 NHANES analysis. However, only two coplanar PCBs and three non-dioxin-like PCB compounds were detected with sufficient frequency to calculate a geometric mean (Exhibit 5-6). Although some PCB congeners were detected with greater frequency during the 2001-2002 survey compared to 1999-2000, this may, in part, be attributed to improved limits of detection in NHANES 2001-2002 (CDC, 2005). After adjusting for a number of covariates (e.g., age, gender, blood cotinine, and lipid level), there were some differences observed in the concentrations of different PCB congeners between different demographic subgroups. However, it is unknown whether these differences represent differences in exposure, pharmacokinetics, or the relationship of dose per body weight (CDC, 2005).

Indicator Limitations

- Because the data from NHANES 1999-2000 and 2001-2002 represent only two survey periods, changes in estimates between the two time periods do not necessarily reflect a trend. As CDC releases additional survey results (e.g., 2003-2004), it will become possible to more fully evaluate trends (CDC, 2002, 2004).
- Generally recognized reference levels for organochlorine pesticides and dioxin, furan, and PCB congeners in blood have not yet been established.

Data Sources

Data used for this indicator were extracted from the CDC report that presents results of the ongoing National Health and Nutrition Examination Survey (CDC, 2005). The underlying laboratory data supporting CDC's report are available online in SAS® transport file format at http://www.cdc.gov/nchs/about/major/nhanes/datalink.htm.

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INDICATOR | Urinary Pesticide Level

Pesticides are chemicals or biological agents that kill plant or animal pests. They include herbicides, insecticides, fungicides, and rodenticides. More than a billion pounds of pesticides are used in the U.S. each year to control weeds, insects, and other organisms that threaten or undermine human activities (Aspelin, 2003). Some of these compounds can be harmful to humans if ingested, inhaled, or otherwise contacted in sufficient quantities. The primary routes of exposure for the general population are ingestion of a treated food source and contact with applications in or near residential sites. Herbicide exposure can also result from contaminated water. Those who manufacture, formulate, and/or apply these chemicals can also be occupationally exposed.

This indicator reports the results of human biomonitoring for three classes of non-persistent insecticides and three classes of herbicides, which can be measured through metabolites that result from the chemical breakdown of the pesticide within the body. Measurement of non-persistent pesticide metabolites in urine typically reflects recent exposure (i.e., in the last few days) due to the short time these metabolites remain within the body (CDC, 2005).

The three classes of insecticides covered by this indicator are carbamates, organophosphates, and pyrethroids. Carbamate insecticides have a wide variety of uses, which include applications on agricultural crops, residential lawns and gardens, and golf courses. Carbamate insecticides do not persist long in the environment, so they have a low potential for bioaccumulation. Organophosphates are used to control a broad spectrum of insects. Although organophosphates are still used for insect control on many food crops, most residential uses are being phased out in the U.S. Pyrethroids are synthetic analogues of pyrethrins, which are natural chemicals found in chrysanthemum flowers. All three groups are neurotoxicants that act by overstimulating the nervous systems of exposed organisms. Symptoms of exposure to pesticides in these classes include muscle weakness or paralysis, difficulty breathing, difficulty concentrating, impaired coordination, and memory loss (CDC, 2005).

The three herbicide classes discussed here are licensed for both commercial and restricted use. Restricted use products can only be applied by certified applicators or under the supervision of such an applicator (U.S. EPA, 2003). The

Exhibit 5-7. Urine concentrations of selected carbamate pesticide metabolites for the U.S. population age 6-59 years, 1999-2002

	Survey years	Sample size	Geometric mean and selected percentiles for carbamate metabolite concentrations[a,b,c]				
			Geometric mean	50th	75th	90th	95th
1-Naphthol[d]							
µg/L of urine	1999-2000	1,998	1.70	1.22	2.72	6.20	12.0
µg/g of creatinine	1999-2000	1,998	1.52	1.25	3.00	6.80	11.6
2-Isopropoxyphenol							
µg/L of urine	1999-2000	1,917	NC	<LOD	<LOD	<LOD	<LOD
	2001-2002	2,503	NC	<LOD	<LOD	<LOD	<LOD
µg/g of creatinine	1999-2000	1,917	NC	<LOD	<LOD	<LOD	<LOD
	2001-2002	2,502	NC	<LOD	<LOD	<LOD	<LOD
Carbofuranphenol							
µg/L of urine	1999-2000	1,994	NC	<LOD	<LOD	<LOD	0.74
	2001-2002	2,530	NC	<LOD	<LOD	<LOD	<LOD
µg/g of creatinine	1999-2000	1,994	NC	<LOD	<LOD	<LOD	0.78
	2001-2002	2,529	NC	<LOD	<LOD	<LOD	<LOD

[a]NC = not calculated; the proportion of results below the limit of detection was too high to provide a valid result.

[b]<LOD = below the limit of detection (LOD) of the analytical method (see CDC, 2005, for chemical-specific LODs).

[c]Refer to CDC, 2005, for confidence intervals for reported values.

[d]1-Naphthol was not included in CDC, 2005.

Data source: *CDC, 2003, 2005*

Exhibit 5-8. Urine concentrations of selected organophosphate pesticide metabolites for the U.S. population age 6-59 years, 1999-2002

			Geometric mean and selected percentiles or organophosphate pesticide metabolite concentrations[a,b,c]				
	Survey years	Sample size	Geometric mean	50th	75th	90th	95th
Dimethylphosphate							
µg/L of urine	1999-2000	1,949	NC	0.74	2.80	7.90	13.0
	2001-2002	2,519	NC	<LOD	3.25	8.22	13.4
µg/g of creatinine	1999-2000	1,949	NC	0.81	2.93	8.46	16.1
	2001-2002	2,518	NC	<LOD	3.00	7.83	12.7
Dimethylthiophosphate							
µg/L of urine	1999-2000	1,948	1.82	2.70	10.0	38.0	46.0
	2001-2002	2,518	NC	0.45	4.02	16.2	32.6
µg/g of creatinine	1999-2000	1,948	1.64	2.12	9.57	32.0	51.0
	2001-2002	2,517	NC	0.85	3.79	13.2	27.2
Dimethyldithiophosphate							
µg/L of urine	1999-2000	1,949	NC	<LOD	2.30	12.0	19.0
	2001-2002	2,518	NC	<LOD	0.89	2.49	4.95
µg/g of creatinine	1999-2000	1,949	NC	<LOD	1.86	10.1	21.7
	2001-2002	2,517	NC	<LOD	0.67	2.60	5.80
Diethylphosphate							
µg/L of urine	1999-2000	1,949	1.03	1.20	3.10	7.50	13.0
	2001-2002	2,520	NC	<LOD	2.76	6.33	11.4
µg/g of creatinine	1999-2000	1,949	0.92	0.92	2.73	7.94	12.1
	2001-2002	2,519	NC	<LOD	2.39	5.23	8.53
Diethylthiophosphate							
µg/L of urine	1999-2000	1,949	NC	0.49	0.76	1.30	2.20
	2001-2002	2,519	0.46	0.57	1.48	2.46	3.94
µg/g of creatinine	1999-2000	1,949	NC	0.25	0.71	1.70	2.64
	2001-2002	2,518	0.45	0.52	1.33	2.84	4.61
Diethyldithiophosphate							
µg/L of urine	1999-2000	1,949	NC	0.08	0.20	0.47	0.87
	2001-2002	2,516	NC	<LOD	<LOD	0.61	0.83
µg/g of creatinine	1999-2000	1,949	NC	0.07	0.20	0.55	0.86
	2001-2002	2,515	NC	<LOD	<LOD	0.58	1.01

[a]NC = not calculated; the proportion of results below the limit of detection was too high to provide a valid result.

[b]<LOD = below the limit of detection (LOD) of the analytical method (see CDC, 2005, for chemical-specific LODs).

[c]Refer to CDC, 2005, for confidence intervals for reported values.

Data source: *CDC, 2005*

Exhibit 5-9. Urine concentrations of selected pyrethroid pesticide metabolites for the U.S. population age 6-59 years, 2001-2002

	Survey years	Sample size	Geometric mean and selected percentiles of pyrethroid pesticide metabolite concentrations[a,b,c]				
			Geometric mean	50th	75th	90th	95th
4-Fluoro-3-phenoxybenzoic acid							
µg/L of urine	2001-2002	2,539	NC	<LOD	<LOD	<LOD	<LOD
µg/g of creatinine	2001-2002	2,538	NC	<LOD	<LOD	<LOD	<LOD
cis-3-(2,2-Dichlorovinyl)-2,2-dimethylcyclopropane carboxylic acid							
µg/L of urine	2001-2002	2,539	NC	<LOD	0.16	0.49	0.89
µg/g of creatinine	2001-2002	2,538	NC	<LOD	0.22	0.44	0.78
trans-3-(2,2-Dichlorovinyl)-2,2-dimethylcyclopropane carboxylic acid							
µg/L of urine	2001-2002	2,525	NC	<LOD	0.41	1.20	2.50
µg/g of creatinine	2001-2002	2,524	NC	<LOD	0.72	1.45	2.55
cis-3-(2,2-Dibromovinyl)-2,2-dimethylcyclopropane carboxylic acid							
µg/L of urine	2001-2002	2,539	NC	<LOD	<LOD	<LOD	<LOD
µg/g of creatinine	2001-2002	2,538	NC	<LOD	<LOD	<LOD	<LOD
3-Phenoxybenzoic acid							
µg/L of urine	2001-2002	2,539	0.32	0.28	0.69	1.69	3.32
µg/g of creatinine	2001-2002	2,538	0.32	0.28	0.58	1.46	3.10

[a]NC = not calculated; the proportion of results below the limit of detection was too high to provide a valid result.

[b]<LOD = below the limit of detection (LOD) of the analytical method (see CDC, 2005, for chemical-specific LODs).

[c]Refer to CDC, 2005, for confidence intervals for reported values.

Data source: *CDC, 2005*

herbicide groups are chlorphenoxy acids, triazines, and chloroacetanilides. Symptoms of acute high-dose exposure to these herbicides can include skin and mucosal irritation as well as burning sensations in the nasopharynx and chest if inhaled (Reigart and Roberts, 1999).

This indicator presents pesticide urinary metabolite data collected as part of the Centers for Disease Control and Prevention's (CDC's) National Health and Nutrition Examination Survey (NHANES). NHANES is a series of surveys conducted by CDC's National Center for Health Statistics that is designed to collect data on the health and nutritional status of the civilian, non-institutionalized U.S. population using a complex, stratified, multistage, probability-cluster design. CDC's National Center for Environmental Health conducted the laboratory analyses for the biomonitoring samples. Beginning in 1999, NHANES became a continuous and annual national survey; biomonitoring for certain environmental chemicals also was implemented. Data for 1999-2000 and 2001-2002 are presented here as a baseline, with the intent of reporting trends over larger time periods in the future. Carbamates, organophosphates, and herbicides were measured as part of NHANES 1999-2000; urinary levels of pyrethroids were added during the NHANES 2001-2002 survey. This indicator presents data for a subsample of survey participants age 6 to 59 years. NHANES also measured levels of a class of persistent pesticides, the organochlorine pesticides, which are not discussed here but can be found under the Blood Persistent Organic Pollutants Level indicator (p. 5-15).

What the Data Show

Carbamates

Exhibit 5-7 presents the geometric means and four percentile values for unadjusted and creatinine-adjusted urinary concentrations of the carbamate pesticide metabolites. Of the three metabolites presented, only 1-naphthol was detected with sufficient frequency to calculate a geometric mean, which was 1.70 micrograms per liter (µg/L) and 1.52 micrograms per gram (µg/g) (creatinine-adjusted).

Organophosphates

NHANES 1999-2000 and 2001-2002 measured urinary concentrations of dialkyl phosphates, which are the primary

metabolites of many organophosphate compounds. Exhibit 5-8 presents the geometric means and four percentile values for urinary concentrations and creatinine-adjusted urinary concentrations of these metabolites. Only three of the six urinary dialkyl phosphates presented (dimethylthiophosphate, diethylphosphate, and diethylthiophosphate) were measured with sufficient frequency above the limit of detection to calculate a geometric mean. The geometric means for those metabolites were 1.82 µg/L (1.64 µg/g creatinine), 1.03 µg/L (0.92 µg/g creatinine), and 0.46 µg/L (0.45 µg/L creatinine), respectively.

Pyrethroids

Pyrethroid (parent and metabolite) compounds were not included in the NHANES 1999-2000 list of analytes measured in urine. During the 2001-2002 NHANES, however, five pyrethroid urinary metabolites were measured in urine samples from a subgroup of participants. Only one of these metabolites, 3-phenoxybenzoic acid, was measured with sufficient frequency above the limit of detection to calculate a geometric mean. The geometric mean concentration of this metabolite measured in urine was 0.32 µg/L (Exhibit 5-9).

Herbicides

During the 1999-2000 survey, none of the direct metabolites of the three primary classes of herbicide were detected in urine with sufficient frequency above the limit of detection to calculate a geometric mean; therefore, data are not displayed. The metabolites 2,4,5-trichlorophenoxyacetic acid and atrazine mercapturate were detected in only 1.2 percent and 3.3 percent, respectively, of the subsample (CDC, 2003). The minor metabolite 2,4-dichlorophenol had a geometric mean of 1.1 µg/L measured in urine; however, this metabolite can also be a result of metabolism of several other chemicals or a byproduct in the manufacture of chemicals. The findings from the 2001-2002 survey were generally consistent with earlier findings showing these metabolites to be frequently near or below the limits of detection. Unlike the 1999-2000 results, 2,4-dichlorophenol samples collected during 2001-2002 were not detected with sufficient frequency above the detection limit to calculate a geometric mean. However, the reported concentrations of this metabolite at the 75th, 90th, and 95th percentile were higher during the 2001-2002 survey than during the 1999-2000 survey (CDC, 2005). (Data not shown.)

Indicator Limitations

- Because the data from NHANES 1999-2000 and 2001-2002 represent only two survey periods, changes in estimates between the two time periods do not necessarily reflect a trend. As CDC releases additional survey results (e.g., 2003-2004) it will become possible to more fully evaluate trends (CDC, 2002, 2004).
- Urine creatinine concentrations were used to adjust the urinary concentrations of pesticides and metabolites of pesticides and phthalates in subsets of adults participating in NHANES. Traditionally, this approach has been used in population groups without much diversity. However, the inclusion of multiple demographic groups (e.g., children) in NHANES may increase the variability in the urinary creatinine levels when comparing across these different study populations (Barr et al., 2004).
- Generally recognized reference levels for carbamate, organophosphate, herbicide, and pyrethroid metabolites in urine have not yet been established.
- Some metabolites may result from sources other than pesticide exposure. For example, 1-naphthol in the urine may reflect multiple sources of exposure, and is therefore not just an indicator of carbamate pesticide exposure.

Data Sources

Data used for this indicator were extracted from two CDC publications that present results of the ongoing NHANES (CDC, 2003, 2005). The underlying laboratory data supporting CDC's report are available online in SAS® transport file format at http://www.cdc.gov/nchs/about/major/nhanes/datalink.htm.

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INDICATOR | Urinary Pesticide Level *(continued)*

Reigart, J.R., and J.R. Roberts. 1999. Recognition and management of pesticide poisonings. Fifth edition. EPA/735/R-98/003. <http://www.epa.gov/oppfead1/safety/healthcare/handbook/handbook.htm>

U.S. EPA (United States Environmental Protection Agency). 2003. Restricted use products (RUP) report. Accessed March 10, 2005. <http://www.epa.gov/opprd001/rup/>

INDICATOR | Urinary Phthalate Level

Phthalates are industrial chemicals added to many consumer products such as food packaging, plastics (plastic bags, garden hoses, recreational toys, medical tubing, plastic clothes, etc.), adhesives, detergents, personal-care products (such as soap, shampoo, nail polish, etc.), and many others. Exposure can occur through food that has been in contact with phthalate containing packaging, as well as direct contact with products that contain phthalates.

Acute high-dose exposure to di-2-ethylhexyl phthalate, for example, may be associated with mild gastrointestinal disturbances, nausea, and vertigo (U.S. EPA, 2005). Chronic exposure to phthalate compounds has been associated with damage to the liver and testes, cancer, and birth defects in animal studies. However, the extent to which these effects occur in humans is the subject of ongoing research; whether detected levels in humans are a health concern is not yet known (CDC, 2005; Kavlock et al., 2002a-g).

This indicator is based on data collected by the National Health and Nutrition Examination Survey (NHANES). NHANES is a series of surveys conducted by the Centers for Disease Control and Prevention's (CDC's) National Center for Health Statistics that is designed to collect data on the health and nutritional status of the civilian, non-institutionalized U.S. population using a complex, stratified, multistage, probability-cluster design. CDC's National Center for Environmental Health conducted the laboratory analyses for the biomonitoring samples. Beginning in 1999, NHANES became a continuous and annual national survey; biomonitoring for certain environmental chemicals also was implemented. Metabolites of phthalates are measured in urine as a biomarker of phthalate exposure in the population. Data for 1999-2000 and 2001-2002 are presented here as a baseline, with the intent of reporting trends across time as more data become available in the future.

What the Data Show

Exhibit 5-10 presents the geometric means and four percentiles for urinary concentrations and creatinine-adjusted urinary concentrations of 12 selected metabolites of phthalates among a subsample of participants age 6 years and older from the most current NHANES (2001-2002). Seven of the 12 phthalates were also measured in the 1999-2000 survey and are also presented in the table. Mono-ethyl phthalate (the metabolite for diethyl phthalate, an industrial solvent used in many products including those containing fragrances) was the phthalate detected in the highest concentration during both surveys (1999-2000 and 2001-2002), with creatinine-adjusted geometric mean concentrations of 163 and 167 micrograms per gram (μg/g) of creatinine, respectively.

In addition, other phthalate compounds such as mono-n-butyl phthalate (a metabolite for dibutyl phthalate, an industrial solvent used in cosmetics, printing inks, insecticides), mono-benzyl phthalate (a metabolite for benzylbutyl phthalate, an industrial solvent used in adhesives, vinyl flooring, and car care products), and mono-2-ethyl-hexyl phthalate (a metabolite for di-2-ethylhexyl phthalate, used to produce flexible plastics) were detected in urine samples. Mono-cyclohexyl phthalate, mono-n-octyl phthalate, and mono-isononyl phthalate were not measured with sufficient frequency above the limit of detection to calculate a geometric mean for those samples collected between 1999 and 2002.

During the 1999-2000 and 2001-2002 surveys, the geometric mean levels for mono-ethyl phthalate, mono-n-butyl phthalate, mono-benzyl phthalate, and mono-2-ethylhexyl phthalate among specified demographic subgroups were compared after adjustment for the covariates of race/ethnicity, age, gender, and urinary creatinine. For those age 6-11 years compared to the older age groups (12-19 years and 20+ years), urinary mono-ethyl phthalate levels were found to be lower, but urinary mono-butyl, mono-benzyl, and mono-2-ethylhexyl phthalates were higher (CDC, 2005). Females tended to have a higher level than males for mono-ethyl, mono-butyl, and mono-benzyl phthalates. Non-Hispanic blacks had higher levels of mono-ethyl phthalate than non-Hispanic whites or Mexican Americans. (Data not shown.)

Indicator Limitations

- Because the data from NHANES 1999-2000 and 2001-2002 represent only two survey periods, changes in estimates between the two time periods do not necessarily reflect a trend. As CDC releases additional survey results (e.g., 2003-2004), it will become possible to more fully evaluate trends (CDC, 2002, 2004).

Exhibit 5-10. Urine concentrations of selected phthalate metabolites in the U.S. population age 6 years and older, 1999-2002[a]

			Geometric mean and selected percentiles of phthalate metabolite concentrations[b,c,d]				
	Survey years	Sample size	Geometric mean	50th	75th	90th	95th
Mono-methyl phthalate							
µg/L of urine	2001-2002	2,782	1.15	1.50	3.30	6.00	9.80
µg/g of creatinine	2001-2002	2,772	1.08	1.33	2.62	5.00	7.97
Mono-isobutyl phthalate							
µg/L of urine	2001-2002	2,782	2.71	2.60	5.70	11.9	17.9
µg/g of creatinine	2001-2002	2,772	2.53	2.44	4.50	8.02	12.0
Mono-(2-ethyl-5-hydroxyhexyl) phthalate							
µg/L of urine	2001-2002	2,782	20.0	20.1	43.6	91.3	192
µg/g of creatinine	2001-2002	2,772	18.8	16.6	32.3	70.8	147
Mono-(2-ethyl-5-oxohexyl) phthalate							
µg/L of urine	2001-2002	2,782	13.5	14.0	29.6	59.9	120
µg/g of creatinine	2001-2002	2,772	12.6	11.2	21.3	45.1	87.5
Mono-3-carboxypropyl phthalate							
µg/L of urine	2001-2002	2,782	2.75	3.00	5.70	10.0	14.6
µg/g of creatinine	2001-2002	2,772	2.57	2.45	4.07	7.25	11.4
Mono-ethyl phthalate							
µg/L of urine	1999-2000	2,536	179	164	450	1,260	2,840
	2001-2002	2,782	178	169	465	1,230	2,500
µg/g of creatinine	1999-2000	2,536	163	141	360	898	1,950
	2001-2002	2,772	167	147	388	975	1,860
Mono-n-butyl phthalate							
µg/L of urine	1999-2000	2,541	24.6	26.0	51.6	98.6	149
	2001-2002	2,782	18.9	20.4	40.4	73.6	108
µg/g of creatinine	1999-2000	2,541	22.4	21.9	38.9	68.3	97.5
	2001-2002	2,772	17.8	17.4	30.4	52.4	81.3
Mono-benzyl phthalate							
µg/L of urine	1999-2000	2,541	15.3	17.0	35.3	67.1	103
	2001-2002	2,782	15.1	15.7	38.0	80.8	122
µg/g of creatinine	1999-2000	2,541	14.0	13.3	25.1	50.1	77.4
	2001-2002	2,772	14.1	13.5	26.6	55.1	90.4
Mono-cyclohexyl phthalate							
µg/L of urine	1999-2000	2,541	NC	<LOD	<LOD	<LOD	1.00
	2001-2002	2,782	NC	<LOD	<LOD	0.40	0.40
µg/g of creatinine	1999-2000	2,541	NC	<LOD	<LOD	<LOD	3.00
	2001-2002	2,772	NC	<LOD	<LOD	0.59	0.85

See notes at end of table.

Continued

Exhibit 5-10 (continued)**.** Urine concentrations of selected phthalate metabolites in the U.S. population age 6 years and older, 1999-2002[a]

			Geometric mean and selected percentiles of phthalate metabolite concentrations[b,c,d]				
	Survey years	Sample size	Geometric mean	50th	75th	90th	95th
Mono-2-ethylhexyl phthalate							
µg/L of urine	1999-2000	2,541	3.43	3.20	7.60	14.8	23.8
	2001-2002	2,782	4.27	4.10	9.80	22.8	38.9
µg/g of creatinine	1999-2000	2,541	3.12	3.08	5.88	10.8	18.5
	2001-2002	2,772	3.99	3.89	7.94	18.2	32.8
Mono-n-octyl phthalate							
µg/L of urine	1999-2000	2,541	NC	<LOD	<LOD	1.60	2.90
	2001-2002	2,782	NC	<LOD	<LOD	<LOD	<LOD
µg/g of creatinine	1999-2000	2,541	NC	<LOD	<LOD	2.40	3.51
	2001-2002	2,772	NC	<LOD	<LOD	<LOD	<LOD
Mono-isononyl phthalate							
µg/L of urine	1999-2000	2,541	NC	<LOD	<LOD	<LOD	3.50
	2001-2002	2,782	NC	<LOD	<LOD	<LOD	<LOD
µg/g of creatinine	1999-2000	2,541	NC	<LOD	<LOD	<LOD	4.29
	2001-2002	2,772	NC	<LOD	<LOD	<LOD	<LOD

[a]1999-2000 data are not available for mono-methyl phthalate, mono-isobutyl phthalate, mono-(2-ethyl-5-hydroxyhexyl) phthalate, mono-(2-ethyl-5-oxohexyl) phthalate, and mono-3-carboxypropyl phthalate.

[b]NC = not calculated; the proportion of results below the limit of detection was too high to provide a valid result.

[c]<LOD = below the limit of detection (LOD) of the analytical method (see CDC, 2005, for chemical-specific LODs).

[d]Refer to CDC, 2005, for confidence intervals for reported values.

Data source: *CDC, 2005*

- Urine creatinine concentrations were used to adjust the urinary concentrations of phthalates and metabolites of phthalates in subsets of adults participating in NHANES. Traditionally, this approach has been used in population groups without much diversity. However, the inclusion of multiple demographic groups (e.g., children) in NHANES may increase the variability in the urinary creatinine levels when comparing across these different study populations (Barr et al., 2004).
- Differences in the excretion of various phthalates may be due to differences in either exposure or toxicokinetics. The low detection rates for some of the long alkyl chain phthalates metabolites may be due to significantly less metabolism to the monoester metabolite.
- It is unknown whether differences between ages, genders, or races/ethnicities represent differences in exposure, body-size relationships, or metabolism.
- Generally recognized reference levels for phthalate metabolites in urine have not been established.

Data Sources

Data used for this indicator were extracted from the CDC report that presents results of the ongoing NHANES (CDC, 2005). The underlying laboratory data supporting CDC's report are available online in SAS® transport file format at http://www.cdc.gov/nchs/about/major/nhanes/datalink.htm.

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Kavlock, R., K. Boekelheide, R. Chapin, M. Cunningham, E. Faustman, P. Foster, et al. 2002c. NTP Center for the evaluation of risks to human reproduction: Phthalates expert panel report on the reproductive and developmental toxicity of di-isononyl phthalate. Reprod. Toxicol. 16(5):679-708.

Kavlock, R., K. Boekelheide, R. Chapin, M. Cunningham, E. Faustman, P. Foster, et al. 2002d. NTP Center for the evaluation of risks to human reproduction: Phthalates expert panel report on the reproductive and developmental toxicity of di-isodecyl phthalate. Reprod. Toxicol. 16(5):655-678.

Kavlock, R., K. Boekelheide, R. Chapin, M. Cunningham, E. Faustman, P. Foster, et al. 2002e. NTP Center for the evaluation of risks to human reproduction: Phthalates expert panel report on the reproductive and developmental toxicity of di(2-ethylhexyl)phthalate. Reprod. Toxicol. 16(5):529-653.

Kavlock, R., K. Boekelheide, R. Chapin, M. Cunningham, E. Faustman, P. Foster, et al. 2002f. NTP Center for the evaluation of risks to human reproduction: Phthalates expert panel report on the reproductive and developmental toxicity of di-n-butyl phthalate. Reprod. Toxicol. 16(5):489-527.

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5.2.3 Discussion

What These Indicators Say About Trends in Human Exposure to Environmental Contaminants

The biomonitoring indicators presented in this section provide an overall representation of the levels of selected contaminants, or metabolites of contaminants, in human blood and urine across the U.S. population. Measurable levels of many of these contaminants appear in at least some subset of the populations tested. Together, these indicators help us understand the extent to which exposure to individual substances has or has not occurred on a national scale. As stated previously, the presence of a contaminant in human tissue does not by itself mean that the contaminant has caused or will cause adverse effects in that person.

Lead, mercury, cadmium, persistent organic pollutant metabolites, and cotinine were reported at varying levels in sampled blood and the metabolites of pesticides and phthalates in the urine of a subset of those tested. Based on the available data, some notable changes in blood levels were reported over time, primarily for the metals. Compared to historical data collected by the Centers for Disease Control and Prevention (CDC), blood lead levels have been steadily declining since the 1980s. The same general observation is true for blood cotinine (see Section 2.4).

Most blood mercury levels in children and women tested were reported below 5.8 micrograms per liter (µg/L)—levels believed not to be associated with harmful health effects. However, nearly 6 percent of women tested showed blood mercury between 5.8 and 58 µg/L. The latter level is considered a general lower bound for neurological effects in developing fetuses and children of exposed mothers.[9]

Current National Health and Nutrition Examination Survey (NHANES) data sets provide some information about variability of biomarkers across age, gender, race, or ethnicity. Such analysis is only possible, however, for those chemicals frequently measured above the level of detection. For example, blood lead levels are highest among children; cadmium levels

[9] Centers for Disease Control and Prevention. 2005. Third national report on human exposure to environmental chemicals. NCEH publication no. 05-0570. <http://www.cdc.gov/exposurereport/report.htm>

are reported highest in the most recent survey in those 20 years and older. Blood mercury levels are reported for children age 1-5 years and women of child-bearing age only, with the highest levels reported in the latter group. In most cases where disparities are observed, it is unknown whether the differences observed represent differences in exposure, pharmacokinetics (absorption, distribution, metabolism, and excretion), or the relationship of dose per body weight.[10]

Limitations, Gaps, and Challenges

Available national-level data provide information on the general magnitude of exposures that are occurring for this subset of contaminants. Further, they serve as a firm foundation or baseline for future analysis. However, available indicator data answer only a part of the question. At this point in time, most of the biomonitoring indicators alone do not (1) enable an extensive assessment of temporal trends; (2) identify and explain possible differences among some subpopulations; (3) provide information on the geographic distribution of the population of concern, or any particular "hot spots" that may exist; (4) reveal exposure conditions; (5) provide information for all contaminants of potential interest; (6) consider exposure to multiple contaminants; or (7) provide perspective as to whether measured levels are elevated or likely to cause harmful effects. These are the most notable limitations, challenges, and data gaps of EPA interest in answering the question of trends in exposure to environmental contaminants.

Temporal Trends

The relatively short time frame of the indicator data set limits the analysis of temporal trends, but these indicators can serve as a baseline for future analysis. Most of the indicators presented to answer this question reflect data from only one or two NHANES sampling periods (1999-2000 and 2001-2002). Only as additional NHANES reports are released every 2 years will meaningful temporal trend analysis be possible. However, CDC has been monitoring blood lead and cotinine since approximately 1976; for these contaminants, more meaningful temporal trend analysis is possible.

Subgroup Analysis

The adequacy of data for subgroup evaluations varies by indicator. The NHANES data sets presented in this chapter contain a sufficiently large sample size to provide reliable age, gender, race, and ethnicity subgroup analyses. In some cases, however, the numbers of observations were insufficient to meet statistical reliability or confidentiality requirements for reporting estimates for all race or ethnicity categories.[11] The benefits of such analyses have been demonstrated in earlier NHANES subgroup comparisons of blood lead levels (e.g., children age 1-5 years, children living in urban or low-income areas), which have allowed resources to be targeted to higher risk or susceptible populations. However, not all ages are represented for all biomarkers in NHANES. Further, in cases where a small percentage of samples had detectable concentrations of the measured contaminant, subgroup comparisons are impossible or less meaningful.

Geographic Trends

The data currently available do not allow for reliable regional subgroup analyses, because the number of geographic regions sampled each year is relatively small. Although the NHANES sampling scheme is designed to obtain a cross-section of data from various regions across the U.S., the data set is not sufficiently representative to allow inferences about regional levels of the selected biomonitoring indicators.

Exposure Conditions

Biomonitoring data alone do not provide information on when or how exposure to a particular contaminant occurred. Many different exposure scenarios (e.g., acute high exposure versus long-term low-level exposures) can lead to the same concentration measured in the body. The measure does not necessarily identify the source(s) of that contaminant or how a person was exposed (e.g., exposure via drinking water versus food versus inhalation; environmental versus non-environmental source). Biomarkers of exposure integrate exposures across multiple exposure routes. Additional information on ambient conditions would be needed to determine what exposures contribute to concentrations in people's bodies. For example, lead in children's blood may come from exposure to airborne sources, contaminated water or food, or contaminated soil or dust. In addition, some biomarkers are not specific to a particular contaminant, making interpretation of the data and their significance uncertain. Lastly, some environmental contaminants are also produced in trace amounts by normal metabolic processes (e.g., formaldehyde and acetone), so their presence cannot always be attributed to external exposure.[12,13]

Other Environmental Contaminants

There are still many contaminants for which no biomonitoring indicators exist, and others that are simply not feasible to analyze using current technology or data collection methods. For example, although it is possible to measure the amount of radiation that a person is exposed to using a dosimeter, biomarkers are not yet feasible for national estimates of exposure to radon. Similar issues of feasibility exist with other contaminants, including most criteria air pollutants (e.g., ozone, nitrogen dioxide, carbon monoxide, and particulate matter), biological agents (e.g., molds, certain infectious agents such

[10] Centers for Disease Control and Prevention. 2005. Third national report on human exposure to environmental chemicals. NCEH publication no. 05-0570.<http://www.cdc.gov/exposurereport/report.htm>

[11] National Center for Health Statistics. 2006. Health, United States, 2006, with chartbook on trends in the health of Americans. DHHS publication no. 2006-1232. Hyattsville, MD Watson, W.P., and A. Mutti. 2004. Role of biomarkers in monitoring exposures to chemicals: Present position, future prospects. Biomarkers 9(3):211-242.

[12] Watson, W.P., and A. Mutti. 2004. Role of biomarkers in monitoring exposures to chemicals: Present position, future prospects. Biomarkers 9(3):211-242.

[13] Bates, M.N., J.W. Hamilton, J.S. LaKind, P. Langenberg, M. O'Malley, and W. Snodgrass. 2005. Workgroup report: Biomonitoring study design, interpretation, and communication—lessons learned and path forward. Environ. Health Perspect. 113(11):1615-1621.

as bacteria or viruses, and dust mites), byproducts from the disinfection of drinking water (e.g., chlorine or chlorine-containing compounds), and several contaminants commonly found in air and drinking water at Superfund sites (e.g., trichloroethylene and tetrachloroethylene, among others). In many cases, biomonitoring for these contaminants is either cost-prohibitive or not yet technologically feasible. However, biomonitoring methods are constantly evolving. For example, CDC has added a number of environmental contaminants to its biomonitoring efforts, which will be included in future reports. These include arsenic, polybrominated compounds, and perfluorinated compounds (e.g., perfluorooctane sulfonate and perfluorooctanoic acid), among others.[14]

In addition, researchers continue to evaluate whether certain chemicals, referred to as endocrine disruptors, may contribute to adverse health effects in humans and may impact the health of future generations. Information about the magnitude and pattern of human exposure to endocrine disruptors is being collected for only a small subset of chemicals that compose this group (e.g., PCBs, DDT and its metabolites); wider testing will be challenging because there are still many compounds that have not yet been classified as endocrine disruptors, but may someday be identified as such. Moreover, understanding the specific window of vulnerability during different stages of development will be critical in evaluating the potential harmful effects of these chemicals.

Multiple Contaminants

Current biomonitoring indicators do not consider the effects of exposures to multiple contaminants. Specifically, biomarker measurements that are collected in NHANES do not provide any perspective on how different classes of contaminants interact with one another once they enter the body and to what extent associated responses are additive, antagonistic, or synergistic.

Clinical Reference or Comparison Levels

For most available biomonitoring indicators, no general scientific consensus exists as to how to interpret measured levels of contaminants in blood and urine. For example, are measured levels associated with some clinical effect or elevated above some "safe" or "background" level? Tracking trends in exposure over time, combined with trends in ambient measurements and health outcome measurements, is a key part of establishing such reference values. Establishing background or reference ranges (distributions) will help in identifying people with unusually high exposure or the percentage of the populations with contaminant exposures above established levels of concern.

5.3 What Are the Trends in Health Status in the United States?

5.3.1 Introduction

An overarching goal of public health agencies is to increase quality and years of healthy life and to eliminate health disparities. Tracking historical trends in general health status can help identify where interventions have improved the health of a population or where interventions may be needed (e.g., exploring causative factors and preventive measures). For example, a key concern for EPA is what possible environmental exposures could be contributing to the diseases or conditions that are the leading causes of death in the U.S.

The topics covered under this question are broad and not intended to represent specific diseases or conditions related to the environment. Environmental contaminants from air, water, and land can influence the overall health of a nation. As described in Section 5.1, however, many factors other than the environment influence the health of a population, such as socio-demographic attributes, behavioral and genetic risk factors, level of preventive care, and quality of and access to health care. Though no consensus exists on the relative contribution of environmental exposures, tracking overall health in the U.S. provides important context for the next section of this chapter, which examines specific acute and chronic diseases and conditions that may be linked more specifically with exposures to environmental contaminants.

As defined by the World Health Organization, health is a state of complete physical, mental, and social well-being, and not the mere absence of disease or infirmity.[15] The health status of a population can be measured by a wide range of factors: birth and death rates, life expectancy, quality of life, morbidity from specific diseases, risk factors, use of ambulatory care and inpatient care, accessibility of health personnel and facilities, financing of health care, health insurance coverage, and many other factors.[16]

While no single set of measures can completely characterize the health of a large and diverse population, CDC and other health agencies worldwide consistently have viewed life expectancy and mortality data as indicators of overall population health because they represent the cumulative effects of social and physical environmental factors, behavioral and genetic risk factors, and the level and quality of health care. These data include the leading causes of mortality (among both infants and

[14] Department of Health and Human Services. 2003. Candidate chemicals for possible inclusion in future releases of the national report on human exposure to environmental chemicals. Federal Register 68(189):56296-56298. September 30.

[15] World Health Organization. 1946. Preamble to the constitution of the World Health Organization as adopted by the International Health Conference, New York, 19-22 June, 1946; signed on 22 July 1946 by the representatives of 61 states (Official Records of the World Health Organization, no. 2, p. 100) and entered into force on 7 April 1948.

[16] U.S. Department of Health and Human Services. 2000. Healthy people 2010: Understanding and improving health. Second edition. Washington, DC: U.S. Government Printing Office. <http://www.health.gov/healthypeople/>

the general population), which provide a broad perspective on the diseases and conditions that are having the greatest impact on the nation's health. Infant mortality is a particularly useful measure of health status, because it indicates both the current health status of the population and predicts the health of the next generation.[17] It reflects the overall state of maternal health as well as the quality and accessibility of primary health care available to pregnant women and infants.

Tracking health status using such indicators provides information on changing or emerging trends. At the beginning of the 20th century, the population of the U.S. was characterized by a low standard of living, poor hygiene, and poor nutrition; communicable diseases and acute conditions were major causes of most premature deaths. Over the course of the century, public health measures such as improved sanitation and drinking water treatment led to a dramatic decrease in deaths due to infectious diseases and a marked increase in life expectancy. As the population has aged, chronic diseases such as heart disease and cancer have become the leading causes of death.[18] These diseases may require a different approach to prevention, detection, and treatment compared to the infectious and acute illnesses more common in the past.

5.3.2 ROE Indicators

Other agencies such as CDC routinely assess the state of the nation's health. EPA has drawn on the comprehensive data collection efforts and assessments conducted by these agencies in addressing this question. Three indicators are used to assess the trends in health status in the U.S. (Table 5-3). *Life expectancy at birth* is the number of years a newborn would expect to live if that person experienced the mortality schedule existing at the time of birth. *Infant mortality* is the number of infants who die before their first birthday. *General mortality* represents the number of all deaths nationwide and provides information on the leading causes of death. Mortality is also tracked using years of potential life lost, or the number of years "lost" by people in a population who die prematurely of a stated cause. These indicators are interrelated—e.g., declines in mortality result in increased life expectancy, and shifts in life expectancy are often used to describe changes in mortality; changes in infant mortality are reflected in general mortality as well.

Where possible, the indicators for this question track health status among subpopulations (e.g., by gender, race, ethnicity). Generally, differences in mortality and life expectancy between black and white Americans have been tracked for the past several decades, in some cases as far back as the 1930s. A broader spectrum of race and ethnic group breakdowns is available for these indicators in more recent years, including American Indian/Alaska Native, Asian or Pacific Islander, and Hispanic origin. Subpopulation data are presented to the extent practicable under "What the Data Show" and/or within indicator exhibits.

Table 5-3. ROE Indicators of Trends in Health Status in the United States

National Indicators	Section	Page
General Mortality	5.3.2	5-33
Life Expectancy at Birth	5.3.2	5-35
Infant Mortality	5.3.2	5-36

[17] National Center for Health Statistics. 2001. Healthy people 2000 final review. Hyattsville, MD: Public Health Service. <http://www.cdc.gov/nchs/data/hp2000/hp2k01-acc.pdf>

[18] Ibid.

Overall mortality is a key measure of health in a population. Three measures of mortality are "all cause" mortality, cause-specific mortality, and years of potential life lost (YPLL). "All cause" mortality counts the total number of deaths due to any cause within a specified year, whereas cause-specific mortality statistics count the number of deaths due to a particular cause in a specified year. YPLL is defined as the number of years between the age at death and a specified age; that is, the total number years "lost" by persons in the population who die prematurely of a stated cause. Ranking the causes of death can provide a description of the relative burden of cause-specific mortality (NCHS, 2005).

This indicator is based on mortality data recorded in the National Vital Statistics System, which registers virtually all deaths nationwide from death certificate data. YPLL is calculated by subtracting the age at death from a selected age (e.g., 65, 75, 85), then summing the individual YPLLs across each cause of death (CDC, 2007). Sixty-five was selected as the age for this indicator to focus on deaths more likely to be attributable to preventable causes and less influenced by increasing age. The temporal coverage of the data is from 1933 to 2004 and data are collected from all 50 states and the District of Columbia.

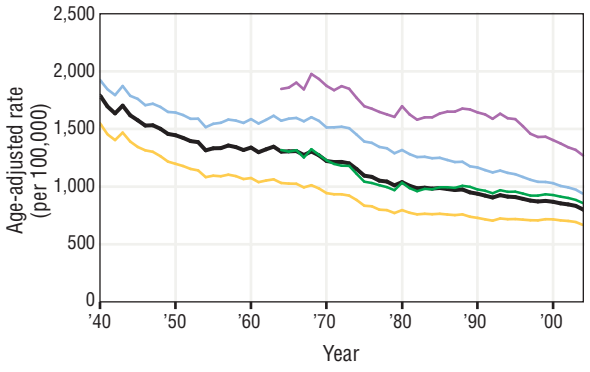
What the Data Show

An increase in the number of deaths in the U.S. has been observed over the last few decades, reflecting the increase in the size and aging of the population. However, the age-adjusted all cause mortality rates have declined yearly since 1980 (except in years of influenza outbreaks in 1983, 1985, 1988, 1993, and 1999) with the most recent available rate of 800.8 deaths per 100,000 people in 2004. Exhibit 5-11 provides some historical perspective on trends in the age-adjusted mortality rates between 1940 and 2003, showing that age-adjusted rates were nearly twice as high in 1940 as they were in 2000. The largest decline in "all cause" mortality rates since 1990 has occurred among black males compared with white males and black and white females.

The rank order of the leading causes of death has remained generally the same since 1999. The one difference is Alzheimer's disease, which was the eighth leading cause of death between 1999 and 2003 but became the seventh leading cause in 2004, displacing influenza and pneumonia. Exhibits 5-12 and 5-13 present the leading causes of mortality and YPLL for 2004, respectively. The three leading causes of death were heart disease, cancer, and stroke, accounting for about 60 percent of all deaths. The YPLL ranking is different, with unintentional injuries, cancer, and heart disease as the leading three causes.

During 2004, heart disease was the leading cause of death across the reported racial and ethnic groups, except for Asians or Pacific Islanders for whom cancer (malignant neoplasms) was the leading cause of death. In addition, diabetes was ranked as the fourth leading cause of death among blacks and American Indians/Alaska Natives (both sexes), which was a higher ranking than for most of the other racial and ethnic groups. (Data not shown.)

Exhibit 5-11. Age-adjusted "all cause" mortality rates in the U.S., 1940-2004[a,b]



[a]Rates are age-adjusted to the 2000 U.S. standard population.

[b]Mortality rates were not generally reported for black males and black females prior to 1964.

Data source: *NCHS, 2001, 2007*

Indicator Limitations

- Cause of death rankings denote the most frequently occurring causes of death among those causes eligible to be ranked. The rankings do not necessarily denote the causes of death of greatest public health importance. Further, rankings of cause-specific mortality could change depending on the defined list of causes that are considered and, more specifically, the types of categories and subcategories that are used for such rankings (NCHS, 2005).
- Mortality rates are based on underlying cause of death as entered on a death certificate by a physician. Incorrect coding and low rates of autopsies that confirm the cause of death may occur. Additionally, some individuals may have had competing causes of death. "When more than one cause or condition is entered by the physician, the underlying cause is determined by the sequence of conditions on the certificate, provisions of the ICD [International Classification of Diseases], and associated selection rules and modifications" (CDC, n.d.). Consequently, some misclassification of reported mortality might occur as a result of these uncertainties, as well as the underreporting of some causes of death.

Data Sources

Mortality rates were obtained from vital statistics reports published by CDC's National Center for Health Statistics (NCHS, 2001, 2007). Data in the NCHS reports are based

in part on unpublished work tables, available on the NCHS Web site at http://www.cdc.gov/nchs/deaths.htm. Leading cause of death and YPLL data were extracted from CDC's Web-Based Injury Statistics Query and Reporting System (WISQARS) (CDC, 2007) (http://www.cdc.gov/ncipc/wisqars/). The underlying data in WISQARS come from CDC/NCHS annual mortality data files.

References

CDC (Centers for Disease Control and Prevention). 2007. National Center for Injury Prevention and Control. Web-Based Injury Statistics Query and Reporting System (WISQARS) [online]. Leading causes of death and years of potential life lost (YPLL) reports, 1999–2004. Accessed October 2, 2007. <http://webappa.cdc.gov/sasweb/ncipc/leadcaus.html> <http://webappa.cdc.gov/sasweb/ncipc/ypll10.html>

CDC. n.d. CDC WONDER: Help page for compressed mortality file. Accessed October 2007. <http://wonder.cdc.gov/wonder/help/cmf.html>

NCHS (National Center for Health Statistics). 2007. Deaths: Final data for 2004. National Vital Statistics Reports 55(19). <http://www.cdc.gov/nchs/data/nvsr/nvsr55/nvsr55_19.pdf>

NCHS. 2005. Deaths: Leading causes for 2002. National Vital Statistics Reports 53(17). <http://www.cdc.gov/nchs/data/nvsr/nvsr53/nvsr53_17.pdf>

NCHS. 2001. Age-adjusted death rates; trend data based on the year 2000 standard population. National Vital Statistics Reports 49(9). <http://www.cdc.gov/nchs/data/nvsr/nvsr49/nvsr49_09.pdf>

Exhibit 5-12. Leading causes of death in the U.S., 2004

Cause of death	Number of deaths	Percent of all deaths[a]
Heart disease	652,486	27.2
Cancer (malignant neoplasms)	553,888	23.1
Stroke (cerebrovascular)	150,074	6.3
Chronic lower respiratory diseases	121,987	5.1
Accidents (unintentional injuries)	112,012	4.7
Diabetes mellitus	73,138	3.1
Alzheimer's disease	65,965	2.8
Influenza and pneumonia	59,664	2.5
Nephritis	42,480	1.8
Septicemia	33,373	1.4
All other causes	532,548	22.2

[a]Totals may not add to 100% due to rounding.

Data source: *CDC, 2007*

Exhibit 5-13. Years of potential life lost (YPLL) before age 65 in the U.S., 2004

Cause of death	YPLL	Percent of all YPLL[a]
Accidents (unintentional injuries)	2,219,044	19.1
Cancer (malignant neoplasms)	1,877,690	16.2
Heart disease	1,413,158	12.2
Perinatal period	922,191	7.9
Suicide	687,395	5.9
Homicide	565,979	4.9
Congenital anomalies	486,853	4.2
HIV	261,784	2.3
Stroke (cerebrovascular)	245,074	2.1
Liver disease	231,132	2.0
All other causes	2,702,330	23.3

[a]Totals may not add to 100% due to rounding.

Data source: *CDC, 2007*

INDICATOR | Life Expectancy at Birth

Life expectancy at birth is often used to appraise the overall health of a given population (NCHS, 2006a). Changes in life expectancy over time are commonly used to describe trends in mortality. Life expectancy is the average number of years at birth a person could expect to live if current mortality trends were to continue for the rest of that person's life.

This indicator is based on data from the National Vital Statistics System, which registers virtually all deaths and births nationwide. The temporal coverage of the data is from 1933 to 2004 and data are collected from all 50 states and the District of Columbia.

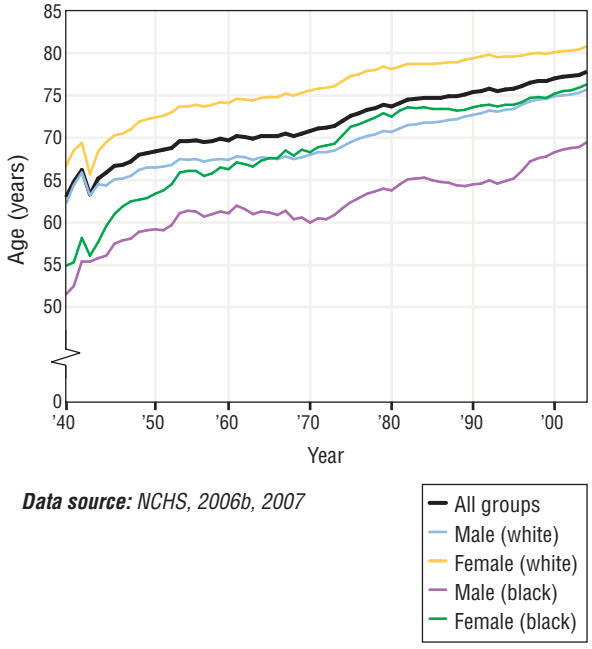
What the Data Show

Exhibit 5-14 presents the historical trends in life expectancy at birth for the entire population as well as by gender and race (black and white) between 1940 and 2004, showing an upward trend in life expectancy in the U.S. over time. Life expectancy at birth has increased throughout the 20th and now into the 21st century. The overall life expectancy was the highest ever reported in 2004 at 77.8 years, increasing from 77.4 in 2003.

Life expectancy continues to increase for both males (73.9 years in 1999 to 75.2 years in 2004) and females (79.4 years in 1999 to 80.4 years in 2004). The gap in life expectancy between males and females widened from 2.0 years to 7.8 years between 1900 and 1979. Recently, this gap narrowed for the year 2000 (a difference of 5.4 years between males and females) and remained relatively constant through 2004 (a difference of 5.2 years between males and females). (Data not shown.)

The increase in life expectancy among blacks reported for 1999 (71.4 years) continued, with a reported life expectancy of 73.1 years in 2004. The difference in life expectancy between the black and white populations was 5.2 years in 2004. In 2004, white females continued to have the highest life expectancy at 80.8 years, followed by black females at 76.3 years, white males at 75.7 years, and black males at 69.5 years (Exhibit 5-14).

Exhibit 5-14. Life expectancy in the U.S. by race and sex, 1940-2004



Data source: *NCHS, 2006b, 2007*

Indicator Limitations

- Life expectancy at birth is strongly influenced by infant and child mortality rates. It is important to consider such influences when making comparisons among subgroups, since differences in life expectancy among certain subgroups may be mostly attributed to differences in prenatal care and other important determinants of infant and child mortality.

Data Sources

The annual life expectancy data used for this indicator were obtained from life tables published by CDC's National Center for Health Statistics (NCHS, 2006b). NCHS also publishes life expectancy data in its annual "deaths: final data" reports (e.g., NCHS, 2007); however, these reports generally provide year-by-year breakdowns beginning in 1975. NCHS life table reports provide annual data back to before 1940. Life table methodologies used to calculate life expectancies are presented in each of these NCHS reports.

References

NCHS (National Center for Health Statistics). 2006a. Health, United States, 2006, with chartbook on trends in the health of Americans. DHHS Publication No. 2006-1232. Hyattsville, MD.

NCHS. 2006b. United States life tables, 2003. National Vital Statistics Reports 54(14). Table 12. <http://www.cdc.gov/nchs/data/nvsr/nvsr54/nvsr54_14.pdf>

NCHS. 2007. Deaths: Final data for 2004. National Vital Statistics Reports 55(19). Table 8. <http://www.cdc.gov/nchs/data/nvsr/nvsr55/nvsr55_19.pdf>

INDICATOR | Infant Mortality

Infant mortality is a particularly useful measure of health status because it both indicates current health status of the population and predicts the health of the next generation (NCHS, 2001). Infant mortality in the U.S. is defined as the death of an infant from time of live birth to the age of 1 year. It does not include still births. Overall infant mortality is composed of neonatal (less than 28 days after birth) and postneonatal (28 days to 11 months after birth) deaths.

This indicator presents infant mortality for the U.S. based on mortality data from the National Vital Statistics System (NVSS) based on death certificate data. The NVSS registers virtually all deaths and births nationwide, with data coverage from 1933 to 2004 and from all 50 states and the District of Columbia.

What the Data Show

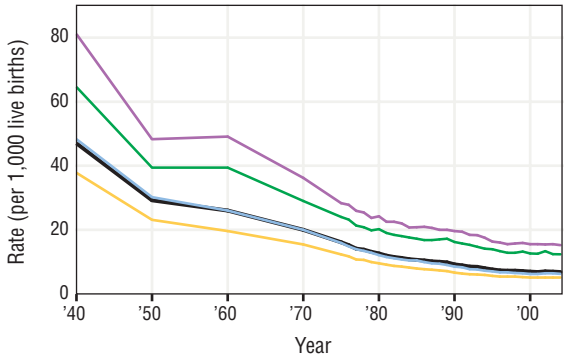
In 2004, a total of 27,936 deaths occurred in children under 1 year of age, 89 fewer deaths than in 2003. Exhibit 5-15 presents the national trends in infant mortality between 1940 and 2004 for all infant deaths as well as infant deaths by gender and race (black and white). A striking decline has occurred during this time period, with overall infant mortality rates dropping from nearly 50 deaths per 1,000 live births in 1940 to just under seven deaths per 1,000 live births in 2004. Beginning around 1960, the infant mortality rate has decreased or remained level each successive year through 2004, except for 2002. From 2000 to 2004, infant mortality rates ranged from 6.8 (2001 and 2004) to nearly 7.0 (2002) per 1,000 live births. Infant mortality rates were highest among black males and lowest among white females, although this gap has been decreasing over time.

The infant mortality rate for blacks decreased from 14.6 per 1,000 live births in 1999 to 13.8 per 1,000 live births in 2004. However, this is still twice the rate compared to white infants, which ranged from approximately 5.7 to 5.8 per 1,000 live births between 1999 and 2004. Infant mortality rates among Hispanic infants have changed little since 1999. In 2004, the infant mortality rate for Hispanic infants was 5.6 per 1,000 live births (NCHS, 2007a). (Data not shown.)

In the U.S. in 2004, the 10 leading causes of infant mortality accounted for nearly 69 percent of all infant deaths, with the subgroup consisting of congenital anomalies (i.e., congenital malformations, deformations, and chromosomal abnormalities) having the highest rate at nearly 1.4 per 1,000 live births. This category alone accounts for approximately 20 percent of all infant deaths in 2004 (Exhibit 5-16).

Congenital anomalies were generally ranked highest among the different racial groups. However, the leading cause of infant mortality among blacks was short gestation and low birthweight, followed by congenital anomalies. There were few differences in the leading causes of infant mortality between Hispanics and non-Hispanics. In addition, the Centers for Disease Control and Prevention (CDC) report a substantial difference in the leading causes of death during the neonatal versus the postneonatal periods. Disorders related to short gestation and low birthweight were the leading cause of death for neonates and sudden infant death syndrome was the leading cause of death for postneonates, based on 2003 data (NCHS, 2007b). (Data not shown.)

Exhibit 5-15. Infant mortality rates in the U.S. by race and sex, 1940-2004[a,b]



[a]Race was reported based on the race of the child (1940-1979) or the race of the mother (1980-2004).

[b]Annual infant mortality rates are not available prior to 1975 in published sources. Trends presented from 1940-1974 are based on data published for 1940, 1950, 1960, and 1970.

Data source: *NCHS, 2007*

Indicator Limitations

- Cause of death rankings denote the most frequently occurring causes of death among those causes eligible to be ranked. The rankings do not necessarily denote the causes of death of greatest public health importance. Further, rankings of cause-specific mortality could change depending on the defined list of causes that are considered and, more specifically, the types of categories and subcategories that are used for such rankings (NCHS, 2005).
- Mortality rates are based on underlying cause of death as entered on a death certificate by a physician. Incorrect coding and low rates of autopsies that confirm the cause of death may occur. Additionally, some individuals may have had competing causes of death. "When more than one cause or condition is entered by the physician, the underlying cause is determined by the sequence of conditions on the certificate, provisions of the ICD [International Classification of Diseases], and associated selection rules and modifications" (CDC, n.d.). Consequently, some misclassification of reported mortality might occur as a result of these uncertainties, as well as the underreporting of some causes of death.

Exhibit 5-16. Leading causes of infant death in the U.S., 2004[a]

Cause of death	Number of deaths	Percent of all infant deaths[b]
Congenital malformations, deformations, and chromosomal abnormalities	5,622	20.1
Disorders related to short gestation and low birthweight	4,642	16.6
Sudden infant death syndrome (SIDS)	2,246	8.0
Newborn affected by maternal complications of pregnancy	1,715	6.1
Accidents (unintentional injuries)	1,052	3.8
Newborn affected by complications of placenta, cord, and membranes	1,042	3.7
Respiratory distress of newborn	875	3.1
Bacterial sepsis of newborn	827	3.0
Neonatal hemorrhage	616	2.2
Circulatory system disease	593	2.1
All other causes	8,706	31.2

[a]"Infant deaths" are those occurring before the age of 1.

[b]Totals may not add to 100% due to rounding.

Data source: *CDC, 2007*

Data Sources

Infant mortality data were obtained from a published report by CDC's National Center for Health Statistics (NCHS, 2007a), which provides annual natality data back to 1975 and decadal data for 1940, 1950, 1960, and 1970. Data in the NCHS report are based in part on unpublished work tables, available on the NCHS Web site at http://www.cdc.gov/nchs/deaths.htm. Leading cause of infant death data were extracted from CDC's Web-Based Injury Statistics Query and Reporting System (WISQARS) (CDC, 2007) (http://www.cdc.gov/ncipc/wisqars/), with supporting documentation from NVSS reports (NCHS, 2007). The underlying data in WISQARS come from CDC/NCHS annual mortality data files.

References

CDC (Centers for Disease Control and Prevention). 2007. National Center for Injury Prevention and Control: Web-Based Injury Statistics Query and Reporting System (WISQARS) [online]. Leading causes of death reports, 1999-2004. Accessed October 8, 2007. <http://webapp.cdc.gov/sasweb/ncipc/leadcaus.html>

CDC. n.d. CDC WONDER: Help page for compressed mortality file. Accessed October 2007. <http://wonder.cdc.gov/wonder/help/cmf.html>

NCHS (National Center for Health Statistics). 2007. Deaths: Final data for 2004. National Vital Statistics Reports 55(19). <http://www.cdc.gov/nchs/data/nvsr/nvsr55/nvsr55_19.pdf>

NCHS. 2005. Deaths: Leading causes for 2002. National Vital Statistics Reports 53(17). <http://www.cdc.gov/nchs/data/nvsr/nvsr53/nvsr53_17.pdf>

NCHS. 2001. Healthy people 2000 final review. Hyattsville, MD: Public Health Service. <http://www.cdc.gov/nchs/data/hp2000/hp2k01-acc.pdf>

5.3.3 Discussion

What These Indicators Say About Trends in Health Status in the United States

ROE indicators used to answer this question show that the overall health of the nation has continued to improve. The three leading causes of death across all age groups—heart disease, cancer, and stroke—remain unchanged since 1999. In contrast, a ranking by years of potential life lost, which weighs deaths at an earlier age more heavily, places unintentional injuries, cancer, and heart disease as the top three (General Mortality indicator, p. 5-33). Although men and women in many other countries have longer life expectancies, general mortality rates in the U.S. continue to decline, and life expectancy continues a long-term upward trend (Life Expectancy indicator, p. 5-35). See Box 5-2 for an overview of health status in the U.S. compared to the rest of the world.

The decline in the all cause mortality rate since 1940 has been driven largely by declines in deaths from heart disease, stroke, and unintentional injuries. These trends have been linked in part to the resources devoted to health education, public health programs, health research, and health care, and the impact of these efforts on controlling disease. For example, public campaigns about smoking and the use of cholesterol-lowering drugs have contributed to a decline in the death rate from heart disease. Efforts to improve motor vehicle safety as well as safety in homes and workplaces have helped to lower death rates from unintentional injuries. New medical treatments have resulted in a decline in the death rate from HIV.[19]

Infant Mortality (p. 5-36), like the other two indicators, shows a long-term decline, likely due to widespread application of advances in medical knowledge (such as the introduction of synthetic surfactant for preterm infants and widespread public education about infant sleep position).[20] However, infant mortality in the U.S. remains among the highest in the industrialized world. In 2003 and 2004, the infant mortality rates decreased after increasing in 2002 for the first time since 1958. The 2002 rise in infant mortality was attributed to an increase in neonatal deaths (infants less than 28 days old), particularly deaths of infants within the first week of life.[21]

Despite a generally improving picture of the nation's health, racial and ethnic disparities in health status persist. For example, though the nation's infant mortality rate has decreased, the infant death rate for black infants is still more than double that of whites. In 2004, the gap in life expectancy between the black and white populations is 5.2 years, though this gap has been narrowing.[22] Differences in death rates also exist between black and white populations. Observed differences are believed to be the result of a complex interaction of genetic variations, environmental factors, and specific health behaviors.[23]

Box 5-2. Worldwide Comparisons in Health Status

The following comparisons are based on the most current statistics for each of the three indicators used to study U.S. health status. The World Health Organization (WHO) calculates its statistics to ensure comparability across data sets; the statistics may not fully match those generated by individual countries and reported in other reports.

Life expectancy: According to the WHO, in 2004, the U.S. ranked 35th in terms of life expectancy for males and females of the 192 WHO member states.[a] Japan reports the highest life expectancy (82 years, compared to the U.S life expectancy of 78 years reported by WHO).

Leading causes of death: The leading causes of death reported in the U.S. continue to be heart disease, cancer, and stroke. Worldwide, as reported for 2002, cardiovascular diseases accounted for the largest percentage of deaths, followed by infectious and parasitic diseases and cancer.[b]

Infant mortality: In 2003, the United States ranked 28th among the 37 countries, territories, cities, or geographic areas with at least 1 million population considered to have completed counts of live births and infant deaths as indicated in the United Nations Demographic Yearbook.[c] The U.S. infant mortality rate for the same time period (6.9 per 1,000 live births) was approximately 2 to 3 times higher than the lowest rates reported worldwide (e.g., in Hong Kong the rate was 2.3, in Singapore 2.5, in Japan 3.0, and in Sweden 3.1, per 1,000 live births).

[a] World Health Organization. 2006. World Health Report. See Statistical Annex Table 1. <http://www.who.int/entity/whr/2006/annex/annex1.xls>

[b] World Health Organization. 2005. Incidence, prevalence, mortality, YLL, YLD and DALYs by sex, cause and region, estimates for 2002 as reported in the World Health Report 2004. <http://www.who.int/healthinfo/bodgbd2002revised/en/index.html>

[c] National Center for Health Statistics. 2006. Health, United States, 2006, with chartbook on trends in the health of Americans. Hyattsville, Maryland. DHHS Publication No. 2006-1232. Table 25. <http://www.cdc.gov/nchs/data/hus/hus05.pdf>

[19] National Center for Health Statistics. 2006. Health, United States, 2006, with chartbook on trends in the health of Americans. DHHS publication no. 2006-1232. Hyattsville, MD. p. 3.

[20] National Center for Health Statistics. 2001. Healthy people 2000 final review. Hyattsville, MD: Public Health Service. p. 206. <http://www.cdc.gov/nchs/data/hp2000/hp2k01-acc.pdf>

[21] National Center for Health Statistics. 2005. Health, United States, 2005, with chartbook on trends in the health of Americans. DHHS publication no. 2005-1232. Hyattsville, MD. p. 66.

[22] Ibid. pp. 11-12.

[23] U.S. Department of Health and Human Services. 2000. Healthy people 2010: Understanding and improving health. Second edition. Washington, DC: U.S. Government Printing Office. <http://www.health.gov/healthypeople/>

Differences also exist between men and women. Based on 2004 data, men have a life expectancy 5.2 years less than that of women and have higher death rates for each of the 10 leading causes of death. However, women have shown increased death rates over the past decade in areas where men have experienced improvements, such as lung cancer.[24]

Limitations, Gaps, and Challenges

The indicators are important and widely accepted measures of population health status. However, the selected indicators cannot be expected to fully answer the question on trends in general U.S. health status. Limitations and information gaps are highlighted here.

The indicators provide a broad measure of health status and include many variables that are not related to the environment. No conclusions, therefore, can or should be drawn about the role of exposure to environmental contaminants using these indicators alone. While declining mortality rates and increasing life expectancy suggest improving health status, these indicators do not address other aspects of health, such as morbidity, perceived well-being, or quality of life.

The use of mortality data presents some limitations, largely related to uncertainties associated with the use of death certificate data. First, correct coding of the underlying cause of death and confirmation by autopsy may not occur. Second, uncertainties in intercensal population estimates can affect conclusions about trends in data sets. In addition, improved data on the health status of population subgroups—particularly across race and ethnic groups—would allow better characterization of potential trends across different groups. Accurate identification of health disparities will require improved data collection and the use of standardized data. For example, problems of race and Hispanic-origin classification can affect Hispanic death rates and the comparison of rates across the Hispanic and non-Hispanic populations.[25]

5.4 What Are the Trends in Human Disease and Conditions for Which Environmental Contaminants May Be a Risk Factor, Including Across Population Subgroups and Geographic Regions?

5.4.1 Introduction

As discussed throughout this report, numerous human diseases and conditions have been linked with exposures to environmental contaminants, some more strongly than others. Identifying diseases that might be associated with environmental contaminants, and determining the existing data sources available for them, is a key part of the effort to better characterize links between environmental exposures and adverse health outcomes.

Tracking overall rates of disease in the nation, independent of exposure, enables the evaluation of disease patterns and emerging trends. It may identify diseases, conditions, and possible risk factors that warrant further study or intervention and can help identify where policies or interventions have been successful. Because the U.S. has a diverse population, an important component of such an analysis is identifying disparities among people of differing races and ethnicities, genders, education and income levels, and geographic locations.

EPA has selected those human diseases and conditions with well-established associations with exposures to environmental contaminants and for which national data are available, recognizing again that in most cases risk factors are multi-factorial and that the development of a particular disease or condition depends on the magnitude, duration, and timing of the exposure. The diseases and conditions addressed in this question are associated with the contaminant sources covered by the questions in the three media chapters (Chapters 2, 3, and 4) of this report. As described in Section 5.1, however, this question is not intended to tie human diseases and conditions to specific changes in the environment being measured at the national level. Covered health outcomes fall into the following five broad categories: cancer, cardiovascular disease, respiratory

[24] National Center for Health Statistics. 2006. Health, United States, 2006, with chartbook on trends in the health of Americans. DHHS publication no. 2006-1232. Hyattsville, MD. pp. 11-12.

[25] National Center for Health Statistics. 2006. Deaths: Final data for 2003. National Vital Statistics Reports 54(13). <http://www.cdc.gov/nchs/data/nvsr/nvsr54/nvsr54_13.pdf>

disease, infectious disease, and birth outcome. The reasons for the inclusion of each are highlighted below.

Cancer

The term "cancer" refers to diseases in which abnormal cells divide without control, losing their ability to regulate their own growth, control cell division, and communicate with other cells. Cancer is the second leading cause of death in the U.S. (General Mortality indicator, p. 5-33). More than one in three people will develop cancer and nearly one in four will die of it.[26,27] In response, scientists continue to explore the role that the exposure to environmental contaminants may play, along with other possible risk factors, in the initiation and development of cancer. Some environmental contaminant exposures are known risk factors for certain types of cancers. Examples include radon and lung cancer and arsenic and skin cancer. Though many types of cancer are suspected of being related to ambient environmental exposures, associations are not always clear because the etiology of cancer is complex and influenced by a wide range of factors. Many factors can increase individual cancer risk, such as age, genetics, existence of infectious diseases, and socioeconomic factors that can affect exposure and susceptibility.

Childhood cancers are dissimilar from cancers in adults and are therefore tracked separately. They affect different anatomic sites and may be of embryonic origin. Though overall cancer incidence rates are lower in children than in adults, childhood cancers are the third leading cause of death in children age 1-19 years.[28] Children may be particularly susceptible to exposures *in utero* or during early childhood because their systems are rapidly developing and affected by evolving hormonal systems.[29] As with many adult cancers, the causes of childhood cancers are unknown for the most part; environmental influences may be a factor and have been the subject of extensive research. Environmental exposures are difficult to evaluate because cancer is rare in children and because of challenges in identifying past exposure levels, particularly during potentially important time periods such as *in utero* or maternal exposures prior to conception.[30]

Cardiovascular Disease

More than one-fourth of the U.S. population lives with a cardiovascular disease, with more than 6 million hospitalizations each year.[31] Coronary heart disease and stroke, two of the major types of cardiovascular disease, rank as the first and third leading causes of death, respectively (General Mortality indicator, p. 5-33), and are leading causes of premature and permanent disabilities. Known risk factors include smoking, high blood pressure, high blood cholesterol, diabetes, physical inactivity, and poor nutrition. Outdoor air pollution and environmental tobacco smoke are also known risk factors for cardiovascular disease. Particulate matter, for example, has been demonstrated to be a likely causal factor in both cardiovascular disease morbidity and mortality. Collective evidence from recent studies suggests excess risk associated with short-term exposures to particulate matter and hospital admissions or emergency department visits for cardiovascular effects.[32,33] Environmental tobacco smoke has been shown to be a risk factor for coronary heart disease morbidity and mortality and may contribute to stroke, though evidence is more limited.[34,35]

Respiratory Disease

Chronic obstructive pulmonary disease (COPD) and asthma are two prevalent chronic respiratory diseases in the U.S. Epidemiological and clinical studies have shown that ambient and indoor air pollution are risk factors in several respiratory health outcomes, including reported symptoms (nose and throat irritation), acute onset or exacerbation of existing disease (e.g., asthma), and deaths.[36,37] The relationship between environmental tobacco smoke and diseases of the respiratory tract has been studied extensively in humans and in animals; environmental tobacco smoke has been shown to produce a variety of upper and lower respiratory tract disorders.[38]

[26] American Cancer Society. 2005. Cancer facts and figures 2005. Atlanta. <http://www.cancer.org/downloads/STT/CAFF2005f4PWSecured.pdf>

[27] National Toxicology Program. 2004. Report on carcinogens. Eleventh edition. U.S. Department of Health and Human Services, Public Health Service. <http://ntp.niehs.nih.gov/ntp/roc/toc11.html>

[28] National Center for Health Statistics. 2004. Deaths: Final data for 2002. National Vital Statistics Reports 53(5). <http://www.cdc.gov/nchs/data/nvsr/nvsr53/nvsr53_05.pdf>

[29] Anderson, L.M., B.A. Diwan, N.T. Fear, and E. Roman. 2000. Critical windows of exposure for children's health: Cancer in human epidemiological studies and neoplasms in experimental animal models. Environ. Health. Perspect. 108(Suppl 3):573-594.

[30] National Cancer Institute. 2005. National Cancer Institute research on childhood cancers. Accessed November 2007. <http://www.cancer.gov/cancertopics/factsheet/sites-types/childhood>

[31] Centers for Disease Control and Prevention. 2005. Preventing heart disease and stroke. Addressing the nation's leading killers—at a glance. Revised August 2005.

[32] U.S. Environmental Protection Agency. 2005. Review of the National Ambient Air Quality Standards for particulate matter: Policy assessment of scientific and technical information. OAQPS Staff Paper.

[33] U.S. Environmental Protection Agency. 2004. Air quality criteria for particulate matter. Volumes I (EPA/600/P-99/002aF) and II (EPA/600/P-99/002bF). National Center for Environmental Assessment—RTP Office, Office of Research and Development.

[34] National Cancer Institute. 1999. Smoking and tobacco control monograph 10: Health effects of exposure to environmental tobacco smoke. <http://cancercontrol.cancer.gov/tcrb/monographs/10/m10_complete.pdf>

[35] U.S. Department of Health and Human Services. 2006. The health consequences of involuntary exposure to tobacco smoke: A report of the Surgeon General. Atlanta, GA. Centers for Disease Control and Prevention, Coordinating Center for Health Promotion, National Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health. <http://www.surgeongeneral.gov/library/secondhandsmoke/report/>

[36] U.S. Environmental Protection Agency. 2005. Review of the National Ambient Air Quality Standards for particulate matter: Policy assessment of scientific and technical information. OAQPS Staff Paper.

[37] U.S. Environmental Protection Agency. 2007. Review of the National Ambient Air Quality Standards for ozone: Policy assessment of scientific and technical information. OAQPS Staff Paper.

[38] State of California. 2005. Proposed identification of environmental tobacco smoke as a toxic air contaminant. Part B: Health effects assessment for environmental tobacco smoke. As approved by the Scientific Review Panel on June 24, 2005. California Environmental Protection Agency, Office of Environmental Health Hazard Assessment. <http://www.arb.ca.gov/regact/ets2006/ets2006.htm>

COPD is a group of diseases characterized by airflow obstruction, resulting in breathing-related symptoms and encompasses chronic obstructive bronchitis and emphysema.[39,40] COPD is the fourth leading cause of death in the U.S. and is the leading cause of hospitalization in U.S. adults, particularly in older adults. It represents a major cause of morbidity, mortality, and disability.[41] Air pollution may be an important contributor to COPD, though approximately 80 to 90 percent of COPD deaths is generally attributed to smoking.[42]

Asthma continues to receive attention in both children and adults. Asthma prevalence increased nearly 74 percent during 1980-1996.[43] During 2001-2003, an average annual 20 million people in the U.S. had asthma.[44] Environmental contaminants such as dust mites, pets, mold, and other allergens are considered important triggers for asthma.[45] In addition, the relationship between environmental tobacco smoke and diseases of the respiratory tract has been studied extensively in humans and in animals; environmental tobacco smoke has been shown to produce a variety of upper and lower respiratory tract disorders.[46]

Infectious Disease

Infectious diseases are acute illnesses caused by bacteria, protozoa, fungi, and viruses. Food and water contaminated with pathogenic microorganisms are the major environmental sources of gastrointestinal illness. Though well-established systems for reporting food- and waterborne cases exist, data reported through these largely voluntary programs must be interpreted with caution because many factors can influence whether an infectious disease is recognized, investigated, and reported. Changes in the number of cases reported could reflect actual changes or simply changes in surveillance and reporting. In addition, many milder cases of gastrointestinal illnesses go unreported or are not diagnosed, making it difficult to estimate the number of people affected every year.

The discovery of bacterial contamination of drinking water as the cause of many cases of gastrointestinal illness represents one of the great public health success stories of the 20th century. Waterborne diseases such as typhoid fever and cholera were major health threats across the U.S. at the beginning of the 20th century. Deaths due to diarrhea-like illnesses, including typhoid, cholera, and dysentery, represented the third largest cause of death in the nation at that time. These types of diarrheal deaths dropped dramatically once scientists identified the bacteria responsible, elucidated how these bacteria were transmitted to and among humans in contaminated water supplies, and developed effective water treatment methods to remove pathogens from water supplies.

In addition to being of food- or waterborne origin, infectious disease can be airborne, arthropod-borne (spread by mosquitoes, ticks, fleas, etc.), or zoonotic (spread by rodents, dogs, cats, and other animals). Legionellosis can be contracted from naturally occurring bacteria found in water and spread through poorly maintained artificial water systems (e.g., air conditioning, ventilation systems). Arthropod-borne diseases, including Lyme disease, Rocky Mountain spotted fever, and West Nile virus, can be contracted from certain ticks and mosquitoes that acquire bacteria or viruses by biting infected mammals or birds.

Birth Outcomes

Birth defects are structural or functional anomalies that present at birth or in early childhood. Birth defects cause physical or mental disability and can be fatal. They affect approximately one out of 33 babies born each year in the U.S. and remain the leading cause of infant mortality (Infant Mortality indicator, p. 5-36). Serious, adverse effects on health, development, and functional ability may be experienced by individuals born with birth defects.[47] Birth defects have been linked with a variety of possible risk factors that can affect normal growth and development—genetic or chromosomal aberrations, as well as environmental factors such as exposure to chemicals; exposure to viruses and bacteria; and use of cigarettes, drugs, or alcohol by the mother. The causes of most birth defects are unknown, but research continues to show the possible influence of environmental exposures (e.g., prenatal exposure to high levels of contaminants such as mercury or PCBs). The relationship between exposure to lower concentrations of environmental contaminants and birth defects, however, is less clear.

Low birthweight delivery and preterm birth are considered important risk factors for infant mortality and birth defects. Low birthweight infants have a significantly increased risk of infant death, and those who survive are more likely to experience long-term developmental disabilities.[48] Multiple birth babies have a low birthweight rate of more than 50 percent,

[39] Mannino, D.M. 2002. COPD epidemiology, prevalence, morbidity and mortality, and disease heterogeneity. Chest 121:121S-126S.

[40] Barnes, P.J. 2000. Chronic obstructive pulmonary disease. Review article. N. Engl. J. Med. 343(4):269-280.

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[42] American Lung Association. 2004. Chronic obstructive pulmonary disease (COPD) fact sheet. Accessed February 7, 2005. <http://www.lungusa.org/site/pp.asp?c=dvLUK9O0E&b=35020>

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[44] Moorman, J.G., R.A. Rudd, C.A. Johnson, M. King, P. Minor, C. Bailey, M.R. Scalia, L.J. Akinbami. 2007. National surveillance for asthma—United States, 1980-2004. In: Surveillance Summaries. MMWR 56(SS08):1-14.

[45] U.S. Institute of Medicine. 2000. Clearing the air. Asthma and indoor air exposures. Washington, DC: National Academy Press.

[46] State of California. 2005. Proposed identification of environmental tobacco smoke as a toxic air contaminant. Part B: health effects assessment for environmental tobacco smoke. As approved by the Scientific Review Panel on June 24, 2005. California Environmental Protection Agency, Office of Environmental Health Hazard Assessment. <http://www.arb.ca.gov/regact/ets2006/ets2006.htm>

[47] Centers for Disease Control and Prevention. 2006. Improved national prevalence estimates for 18 selected major birth defects—United States, 1999-2001. MMWR 54(51&52):1301-1305.

[48] National Center for Health Statistics. 2005. Health, United States, 2005, with chartbook on trends in the health of Americans. DHHS publication no. 2005-1232. Hyattsville, MD. p. 11.

compared to approximately 6 percent among singletons, among whom the low birthweight rate rose only 1 percent from 1989 to 1998.[49] To eliminate the effect that multiple births may have on birth outcomes, this report presents data for singleton births only.

Environmental exposures are being investigated for possible associations with birth outcomes such as low birthweight, preterm births, and infant mortality. Some of the risk factors for low birthweight infants born at term include maternal smoking, weight at conception, and nutrition and weight gain during pregnancy.[50] Specific examples of known or suspected environmental contaminant influences on birth outcomes include environmental tobacco smoke, lead, and air pollution. The most robust evidence exists for environmental tobacco smoke and lead.[51] Environmental tobacco smoke is associated with increased risk of low birthweight, preterm delivery, and sudden infant death syndrome.[52] Several studies have identified lead exposure as a risk factor for preterm delivery.[53] Associations between air pollution and fetal growth and infant mortality have been documented. Recent studies report significant associations between PM_{10} concentration averaged over a month or a trimester of gestation and the risk of intrauterine growth reduction and low birthweight.[54] Growing evidence shows exposure-response relationships between maternal exposures to air pollutants (e.g., sulfur dioxide and particulates) and preterm birth.[55,56] Research continues, however, in establishing causal relationships between air pollution and low birthweight and preterm birth. Researchers also continue to examine possible associations between other contaminants as birth outcome risk factors, such as pesticides, polycyclic aromatic hydrocarbons, and others.

5.4.2 ROE Indicators

EPA has selected indicators of health outcomes for which environmental exposures may be a risk factor and for which nationally representative data are available. Nine indicators were selected to address the question (Table 5-4)—two for cancer (including the leading sites of cancer in adults and children), one for cardiovascular disease (including coronary heart disease, stroke, and hypertension), two related to respiratory disease (including asthma and chronic lung conditions such as bronchitis and emphysema), one for infectious diseases (composed of multiple diseases and conditions), and three for birth outcomes.

Table 5-4. ROE Indicators of Trends in Human Disease and Conditions for Which Environmental Contaminants May Be a Risk Factor

National Indicators	Section	Page
Cancer Incidence	5.4.2	5-43
Childhood Cancer Incidence	5.4.2	5-46
Cardiovascular Disease Prevalence and Mortality (N/R)	5.4.2	5-48
Chronic Obstructive Pulmonary Disease Prevalence and Mortality (N/R)	5.4.2	5-52
Asthma Prevalence	5.4.2	5-55
Infectious Diseases Associated with Environmental Exposures or Conditions	5.4.2	5-59
Birth Defects Prevalence and Mortality	5.4.2	5-62
Low Birthweight	5.4.2	5-65
Preterm Delivery	5.4.2	5-67

N/R = National Indicator displayed at EPA Regional scale

[49] National Center for Health Statistics. 2001. Healthy people 2000 final review. Hyattsville, MD: Public Health Service. p. 208. <http://www.cdc.gov/nchs/data/hp2000/hp2k01-acc.pdf>

[50] U.S. Department of Health and Human Services. 2000. Healthy people 2010: Understanding and improving health. Second edition. Washington, DC: U.S. Government Printing Office. <http://www.health.gov/healthypeople/>

[51] Behrman, R.E., and A. Stith Butler, eds. 2007. Preterm birth: Causes, consequences, and prevention. Committee on Understanding Premature Birth and Assuring Healthy Outcomes. Institute of Medicine of the National Academies. Washington, DC: National Academies Press.

[52] State of California. 2005. Proposed identification of environmental tobacco smoke as a toxic air contaminant. Part B: Health effects assessment for environmental tobacco smoke. As approved by the Scientific Review Panel on June 24, 2005. California Environmental Protection Agency, Office of Environmental Health Hazard Assessment. <http://www.arb.ca.gov/regact/ets2006/ets2006.htm>

[53] Agency for Toxic Substances and Disease Registry. 2005. Toxicological profile for lead (update). Draft for public comment. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service.

[54] U.S. Environmental Protection Agency. 2005. Review of the National Ambient Air Quality Standards for particulate matter: Policy assessment of scientific and technical information. OAQPS Staff Paper.

[55] Behrman, R.E., and A. Stith Butler, eds. 2007. Preterm birth: Causes, consequences, and prevention. Committee on Understanding Premature Birth and Assuring Healthy Outcomes. Institute of Medicine of the National Academies. Washington, DC: National Academies Press.

[56] Sram, R.J., B. Binkova, J. Dejmek, and M. Bobak. 2005. Ambient air pollution and pregnancy outcomes: A review of the literature. Environ. Health Perspect. 113(4):375-382.

The indicators used to answer this question are drawn from the Centers for Disease Control and Prevention's vital statistics and surveillance data, including the CDC WONDER Mortality Database, the Summary of Notifiable Diseases, the National Center for Health Statistics' National Vital Statistics Reports and VitalStats Database, and the National Health Interview Survey, as well as the National Cancer Institute's Surveillance, Epidemiology, and End Results Database. The time frames covered generally range back to the 1970s for mortality and incidence data and to the mid-1990s for prevalence data.

In answering this question, both disease morbidity (incidence or prevalence) and mortality (resulting death) statistics are used. Depending on the health outcome of interest, both measures can provide useful insights about trends in disease. Both morbidity and mortality statistics are influenced by a number of factors, however, such as the accuracy of reporting mechanisms and issues related to access to, quality of, and advances in medical care. An overall understanding of the disease measures and associated statistics used to answer this question is important (see Box 5-3).

Where possible, the indicators provide breakouts of population subgroups, such as race, ethnicity, age, and gender. Subpopulation data are presented to the extent practicable under "What the Data Show," within text or shown in indicator figures. For cardiovascular and respiratory diseases, mortality statistics are provided for each of the 10 EPA Regions. For cancer, data for the most frequently diagnosed cancer sites in adults and children, along with overall cancer rates, are used to answer the question.

Box 5-3. Morbidity and Mortality Measures

Both morbidity and mortality can be measured using occurrences or rates. Occurrences represent frequency counts, while rates enable a comparison across populations. Rates are ratios that calculate the frequency of cases (of disease, condition, outcome) divided by the size of the defined population for a specified time period. Usually some constant (generally a multiplier of the power 10) is applied to convert the rate to a whole number.

Morbidity data are often used to describe the incidence and prevalence of a disease or condition. Both incidence and prevalence are often expressed as a rate per 1,000 persons over a particular time period. "Incidence" refers to the number of new cases of a disease or condition in a population during a specified time period. "Prevalence" refers to the total number of people with a given disease or condition in a population at a specified point in time.

Mortality is generally expressed as a rate and is defined as the proportion of the population who die of a disease or condition during a specified time period. The rate is usually calculated for a calendar year and is often expressed per 100,000 persons.

Incidence, prevalence, and mortality statistics can be used to compare the rates of disease at two or more points in time, across different populations (ages, gender, racial/ethnic groups), or between different geographic areas. In general, disease incidence, prevalence, and mortality increase with age. For this reason, when comparing different populations, the data must be adjusted to account for the age differences between the populations. The adjusted data, called "age-adjusted rates," are used where possible in answering this question. Age-adjusted rates are weighted sums of age-specific rates and calculated using standard population factors. (In this report, the 2000 U.S. standard population was used.) Unadjusted rates are referred to as "crude" rates.

INDICATOR | Cancer Incidence

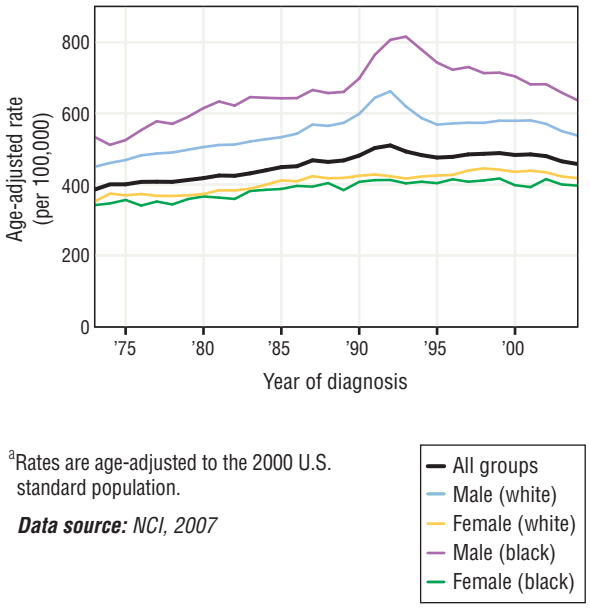
The term "cancer" is used to characterize diseases in which abnormal cells divide without control. A cancerous cell loses its ability to regulate its own growth, control cell division, and communicate with other cells. Cancer cells can invade nearby tissues and can spread through the bloodstream and lymphatic system to other parts of the body (NCI, n.d.). The risk of developing cancer increases with age. Environmental exposures, genetic predisposition, certain viruses, and socioeconomic factors may all play a role in the development and progression of the disease.

For the U.S. population, age-adjusted cancer incidence rates for all sites combined have been stable since 1992 (Edwards et al., 2005). Nevertheless, cancer continues to be the second leading cause of death in the U.S., accounting for about 23 percent of all deaths in 2004 (General Mortality indicator, p. 5-33) (NCHS, 2007). Many different types of cancer exist. These can develop in various organs and tissues within the body and contributing causal factors can vary depending on the cancer site and type. Therefore, tracking rates for individual cancer sites is more meaningful when evaluating cancer trends.

Many factors are known to contribute, or suspected of contributing, to cancer risk. Factors including individual food and beverage preferences, use of tobacco products, exposure to natural and medical radiation (including sunlight), workplace exposures, and pharmaceutical use as well as exposure to substances in the air, water and soil all may contribute individually (i.e., additively) or synergistically

INDICATOR | Cancer Incidence *(continued)*

Exhibit 5-17. Age-adjusted cancer incidence rates in the U.S., 1973-2004: All cancer sites for all ages, by race and sex[a]



[a]Rates are age-adjusted to the 2000 U.S. standard population.

Data source: *NCI, 2007*

(i.e., producing an effect greater than the sum of each factor acting alone) to the development of cancer (NTP, 2004). Further, the cancer hazard to any individual is dependent on the amount and duration of exposure and the individual's susceptibility to a particular substance. Only in a small number of cases is it known what specific exposures or conditions are responsible for the onset and development of cancers (NTP, 2004).

This indicator presents cancer incidence rates for the U.S. population using data collected through the National Cancer Institute's Surveillance, Epidemiology, and End Results (SEER) Program. The SEER Program collects and publishes cancer incidence and survival data from 14 population-based cancer registries and three supplemental registries covering approximately 26 percent of the U.S. population. The 10 most commonly diagnosed cancer sites presented are based on 2004 data compiled from SEER. Site classifications (e.g., lung and bronchus, colon and rectum) were compared to the American Cancer Society's "leading sites" classification to ensure consistency in how data are presented (ACS, 2004).

What the Data Show

Although a slow steady increase in cancer incidence occurred between 1973 and 1992, peaking in 1992 with an age-adjusted cancer incidence of 510 cases per 100,000,

Exhibit 5-18. Age-adjusted cancer incidence rates in the U.S., 2004: Ten leading cancer sites by sex[a]

Percent of all cancers	Rate[b]	Male	Female	Rate[b]	Percent of all cancers
29.9	159.5	Prostate	Breast	124.3	30.7
13.8	73.6	Lung and bronchus	Lung and bronchus	50.2	12.4
10.6	56.7	Colon and rectum	Colon and rectum	41.7	10.3
6.8	36.3	Urinary bladder	Corpus uteri	23.9	5.9
4.6	24.7	Non-Hodgkin's lymphoma	Non-Hodgkin's lymphoma	17.1	4.2
4.5	24.1	Melanoma of the skin	Melanoma of the skin	16.5	4.1
3.3	17.8	Kidney and renal pelvis	Thyroid	14.4	3.6
2.9	15.5	Oral cavity and pharynx	Ovary	12.6	3.1
2.9	15.4	Leukemia	Pancreas	9.8	2.4
2.5	13.3	Pancreas	Urinary bladder	9.1	2.2
18.2	NC[c]	All other sites	All other sites	NC[c]	21.1

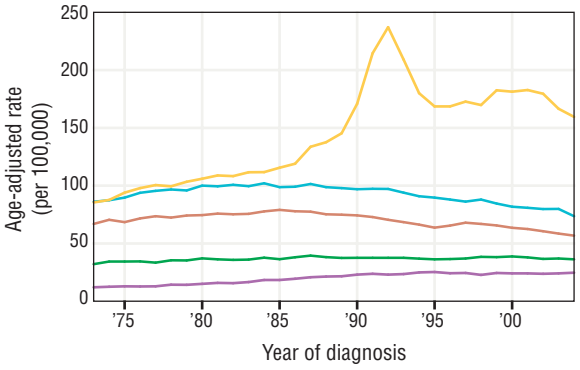
[a]Excludes basal and squamous cell skin cancers and in situ carcinoma, except urinary bladder.

[b]Rates are per 100,000 and age-adjusted to the 2000 U.S. standard population.

[c]NC = not calculated

Data source: *NCI, 2007*

Exhibit 5-19. Age-adjusted cancer incidence rates in the U.S., 1973-2004: Top five cancers in males of all ages[a]

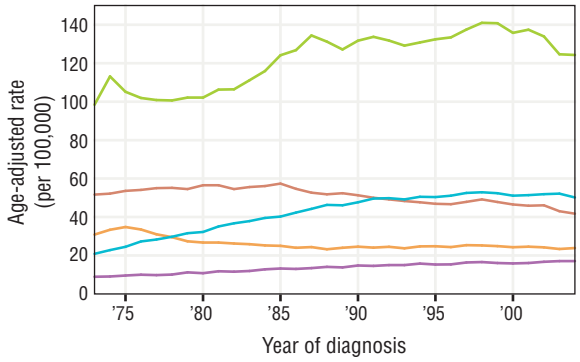


[a]Rates are age-adjusted to the 2000 U.S. standard population.

Data source: *NCI, 2007*

- Colon and rectum
- Lung and bronchus
- Non-Hodgkin's lymphoma
- Prostate
- Urinary bladder

Exhibit 5-20. Age-adjusted cancer incidence rates in the U.S., 1973-2004: Top five cancers in females of all ages[a]



[a]Rates are age-adjusted to the 2000 U.S. standard population.

Data source: *NCI, 2007*

- Breast
- Colon and rectum
- Corpus uteri
- Lung and bronchus
- Non-Hodgkin's lymphoma

overall incidence rates appear to have stabilized over the last 10 years (Exhibit 5-17). Some differences exist in incidence rates across age, gender, and racial groups. During 2004, those age 65 and older had the highest incidence rates (2,102.4 cases per 100,000) compared to all other age categories (data not shown). Total (all sites combined) cancer incidence rates are higher for males compared to females and for black males compared to white males (Exhibit 5-17). The age-adjusted cancer incidence rate in 2004 for black males was 637.2 cases per 100,000 compared to 537.9 cases per 100,000 for white males; among females, the age-adjusted cancer incidence rate in 2004 was 417.9 cases per 100,000 for white females compared to 396.6 cases per 100,000 among black females.

Exhibit 5-18 shows the differences between the top 10 cancer sites in males and females. For both, the top three cancers represent over half of all newly identified cancer cases in 2004. Among the most notable differences is the rate of urinary bladder cancer among males (36.3 cases per 100,000), which is nearly four times that of females (9.1 cases per 100,000). Melanoma of the skin is also higher among males (24.1 cases per 100,000) than females (16.5 cases per 100,000). Thyroid cancer appears as the seventh leading cancer in females (14.4 cancers per 100,000), but is not among the top 10 for males (5.1 cases per 100,000).

Among males, prostate cancer incidence rates increased dramatically between 1986 and the early 1990s, with a decline in rates between 1992 and 1995. This increase is likely due to the introduction of serum prostate-specific antigen testing for the early detection and screening of prostate cancer (Hankey et al., 1999). The other four leading cancers (colon and rectum, lung and bronchus, urinary bladder, and non-Hodgkin's lymphoma) have either been relatively stable or have showed a small decline over the last decade (Exhibit 5-19).

Recent trends (i.e., since 1995) among the less prevalent site-specific cancers in males show small increases in the incidence rates for melanoma of the skin (melanoma), which ranged from 20.2 (1995) to 24.2 (2001) cases per 100,000, and cancers of the kidney and renal pelvis (kidney), which ranged from 15.1 (1997) to 17.8 (2003, 2004) cases per 100,000. Overall, slightly decreasing rates were observed for leukemia, which ranged from 17.6 (1995) to 15.4 (2004) cases per 100,000, and cancers of the oral cavity and pharynx (oral cavity), which ranged from 17.7 (1996) to 15.3 (2001, 2003) cases per 100,000. (Data not shown.)

As shown in Exhibit 5-20, among females, breast cancer remains the leading cancer and rates have generally increased for much of the reporting period. While lung cancer among males has slowly declined over the past decade, the rate among women has generally increased over time and is the second leading cancer among men and women in 2004. The incidence rate of colon cancer among women increased between 1973 and 1985 and has slowly declined since. The incidence of uterine (corpus uteri) cancer in females was relatively stable since 1986, with a small decrease in more recent years, ranging from 25.4 (1997) to 23.3 (2003) cases per 100,000. The incidence rate of non-Hodgkin's lymphoma has exhibited a slow increase since 1973.

INDICATOR | Cancer Incidence *(continued)*

Recent trends in cancer incidence rates among the less prevalent site-specific cancers in females showed increases for melanoma, which ranged from 13.7 (1995) to 16.5 (2004) cases per 100,000 and thyroid cancer, which ranged from 8.9 (1995) to 14.4 (2004) cases per 100,000. Incidence rates decreased for cancers of the ovary, which ranged from 14.7 (1997) to 12.6 (2004) cases per 100,000. (Data not shown.)

Indicator Limitations

- SEER data cover approximately 26 percent of the U.S. population, though it is designed to be representative of the entire U.S. population.
- Incidence data generated from SEER are updated annually. There may be changes in the numerator (e.g., revised counts of newly identified cases) or denominator (i.e., revised population counts) numbers that result in small changes in the overall incidence rates for the same year, depending on when a query is run within the SEER database. For example, the SEER database queried in 2005 generating incidence rates for the year 2000 may provide different incidence rates than the database queried in 2004 for the year 2000.

Data Sources

Cancer incidence data for this indicator were obtained by querying the National Cancer Institute's SEER Program database through the Cancer Query Systems Web-based interface (NCI, 2007), available at http://www.seer.cancer.gov/canques/incidence.html.

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Edwards, K.E., M.L. Brown, P.A. Wingo, H.L. Howe, E. Ward, L. Reis, et al. 2005. Annual report to the nation on the status of cancer, 1975-2002, featuring population-based trends in cancer treatment. J. Natl. Cancer Inst. 97(19):1407-1427.

NCHS (National Center for Health Statistics). 2007. Deaths: Final data for 2004. National Vital Statistics Reports 55(19). <http://www.cdc.gov/nchs/data/nvsr/nvsr55/nvsr55_19.pdf>

NCI (National Cancer Institute). 2007. Surveillance, Epidemiology, and End Results (SEER) Program CANQUES database. SEER registry limited use, Nov 2006, Sub (1973-2004). National Cancer Institute, DCCPS, Surveillance Research Program. Released April 2007, based on November 2006 submission. Accessed September 2007. <http://www.seer.cancer.gov/canques/incidence.html>

NCI. n.d. Dictionary of cancer terms. Accessed October 7, 2004. <http://cancer.gov/dictionary/>

NTP (National Toxicology Program). 2004. Report on carcinogens. Eleventh edition. U.S. Department of Health and Human Services, Public Health Service. <http://ntp.niehs.nih.gov/ntp/roc/toc11.html>

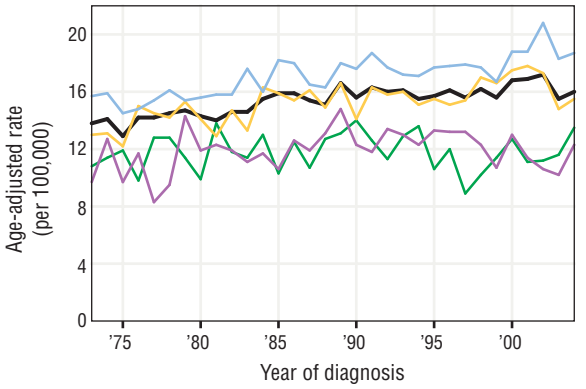
INDICATOR | Childhood Cancer Incidence

The term "cancer" is used to characterize diseases in which abnormal cells divide without control. A cancerous cell loses its ability to regulate its own growth, control cell division, and communicate with other cells. If left unchecked, cancer cells can invade nearby tissues and can spread through the bloodstream and lymphatic system to other parts of the body. The cellular changes caused by cancer cells are complex and occur over a period of time. This may be accelerated in children. The classification of cancers in children differs from the classification used for adult cancers. The International Classification of Childhood Cancer classifies childhood cancer based on tumor morphology rather than, as for adults, the site of the tumor (NCI, 2004).

The causes of childhood cancers are largely unknown. Only a small percentage of cases can be explained by a few conditions such as specific chromosomal/genetic abnormalities (e.g., Down's syndrome) and ionizing radiation exposure (NCI, 2005). Environmental exposures have long been suspected of increasing the risk of certain childhood cancers. Researchers continue to examine environmental influences on childhood cancer (NCI, 2005).

This indicator presents incidence rates for childhood cancers using data collected through the National Cancer Institute's Surveillance, Epidemiology, and End Results (SEER) Program. The SEER Program collects and publishes cancer incidence and survival data from 14 population-based cancer

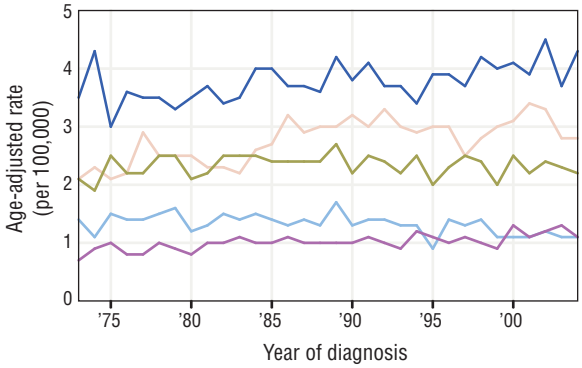
Exhibit 5-21. Age-adjusted cancer incidence rates in the U.S., 1973-2004: All cancer sites for ages 0-19, by race and sex[a]



[a]Rates are age-adjusted to the 2000 U.S. standard population, age 0-19 years.

Data source: *NCI, 2007*

Exhibit 5-22. Age-adjusted cancer incidence rates in the U.S., 1973-2004: Top five cancers for ages 0-19[a]



[a]Rates are age-adjusted to the 2000 U.S. standard population, age 0-19 years.

Data source: *NCI, 2007*

registries and three supplemental registries covering approximately 26 percent of the U.S. population.

What the Data Show

In general, overall childhood (ages 0-19 years) cancer incidence for the U.S. has increased slightly between 1973 and 2004 (Exhibit 5-21), increasing over time from an age-adjusted incidence rate of 13.8 per 100,000 in 1973 to a high of 17.2 per 100,000 in 2002. A rate of 16.0 per 100,000) was reported in 2004. Males generally had higher rates than females, although for some years the reverse was true. Incidence among black females and males age 0-19 years was lower than among white females and males. In 2004, black females and males age 0-19 years had overall incidence rates of 13.5 and 12.3 per 100,000, respectively, compared to white females and males with rates of 15.5 and 18.7 per 100,000 (Exhibit 5-21).

Exhibit 5-22 presents the age-adjusted incidence rates for the top five cancers among children 0-19 years of age between 1973 and 2004. In general, there are no clearly identifiable trends among any of the top five cancers over the reported time period. Leukemia continues to be the most frequently diagnosed cancer in children age 0-19 years.

Indicator Limitations

- SEER data cover approximately 26 percent of the U.S. population, though it is designed to be representative of the entire U.S. population.
- Incidence data generated from SEER are updated annually. There may be changes in the numerator (e.g., revised counts of newly identified cases) or denominator (i.e., revised population counts) numbers that result in small changes in the overall incidence rates for the same year, depending on when a query is run within the SEER database. For example, the SEER database queried in 2005 generating incidence rates for the year 2000 may provide different incidence rates than the database queried in 2004 for the year 2000.

Data Sources

Cancer incidence data for this indicator were obtained by querying the National Cancer Institute's SEER Program database through the Cancer Query Systems Web-based interface (NCI, 2007), available at http://www.seer.cancer.gov/canques/incidence.html.

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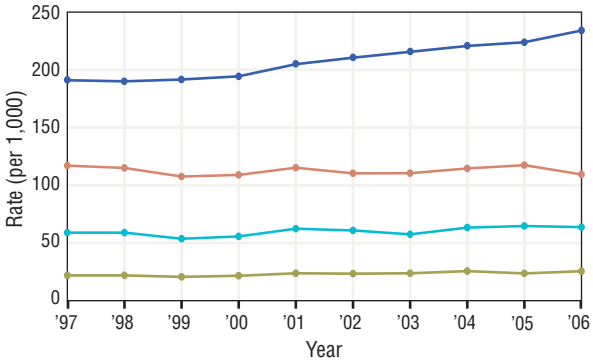
INDICATOR | Cardiovascular Disease Prevalence and Mortality

The broad category of cardiovascular disease (CVD) includes any disease involving the heart and blood vessels. Coronary heart disease, cerebrovascular disease (commonly known as stroke), and hypertension are the major cardiovascular diseases (American Heart Association, 2007). In addition to being a major risk factor for heart disease and stroke, hypertension is a commonly diagnosed disease that can also lead to kidney damage and other health problems. Obesity, physical inactivity, and sodium intake are all important risk factors for hypertension (NIH, 2004). Since 1900, CVD has been the leading cause of death in the U.S. every year except 1918 (American Heart Association, 2007) (General Mortality indicator, p. 5-33). The U.S. age-adjusted mortality rate for CVD reached a peak in 1950 (CDC, 1999). Between 1950 and 1999, the age-adjusted mortality rate for CVD declined 60 percent. The major risk factors for CVD include tobacco use, high blood pressure, high blood cholesterol, diabetes, physical inactivity, and poor nutrition (CDC, 2004; American Heart Association, 2007).

Environmental exposures may also play a role in CVD morbidity and mortality independent of other risk factors. However, susceptible populations such as the elderly and other high-risk populations may be most impacted. For example, studies have shown exposure to ambient airborne particulate matter to be associated with increased hospitalizations and mortality among older individuals, largely due to cardiopulmonary and cardiovascular disease (U.S. EPA, 2004). Environmental tobacco smoke (ETS) may also contribute to CVD. Although the smoke to which a nonsmoker is exposed is less concentrated than that inhaled by smokers, research has demonstrated increased cardiovascular-related health risks associated with ETS (State of California, 2005).

This indicator presents U.S. adult (age 18 and older) prevalence rates for heart disease (all types), coronary heart disease, stroke, and hypertension; and mortality rates for CVD as a whole as well as coronary heart disease (including myocardial infarction), stroke, and hypertension. CVD prevalence data were compiled between 1997 and 2006 from the National Health Interview Survey (NHIS), conducted by the Centers for Disease Control and Prevention's (CDC's) National Center for Health Statistics (NCHS). The NHIS is the principal source of information on the health of the civilian non-institutionalized population of the U.S. and since 1960 has been one of the major data collection programs of NCHS. CVD prevalence is based on the number of adults who reported that they had ever been told by a doctor or other health practitioner that they had a specified CVD. Mortality data (all ages) were compiled between 1979 and 2004 using the National Vital Statistics System (NVSS), maintained by NCHS. The NVSS registers virtually all deaths and births nationwide, with data coverage from 1933 to 2004 and from all 50 states and the District of Columbia.

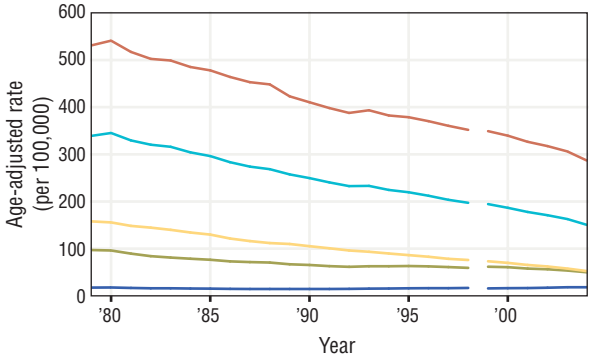
Exhibit 5-23. Cardiovascular disease prevalence in U.S. adults (age 18 and older), 1997-2006[a]



[a]Rates presented are crude rates.

Data source: *NCHS, 1999-2005, 2006a,b, 2007*

Exhibit 5-24. Age-adjusted cardiovascular disease mortality rates in the U.S., 1979-2004[a,b]



[a]Due to differences in the ICD system used for classifying mortality, data from 1979-1998 should not be directly compared to data from 1999-2004 [ICD-9 codes: 390-434, 436-448 (1979-1998); ICD-10 codes: I00-I78 (1999-2004)].

[b]Rates are age-adjusted to the 2000 U.S. standard population.

Data source: *CDC, 2007*

What the Data Show

CVD Prevalence

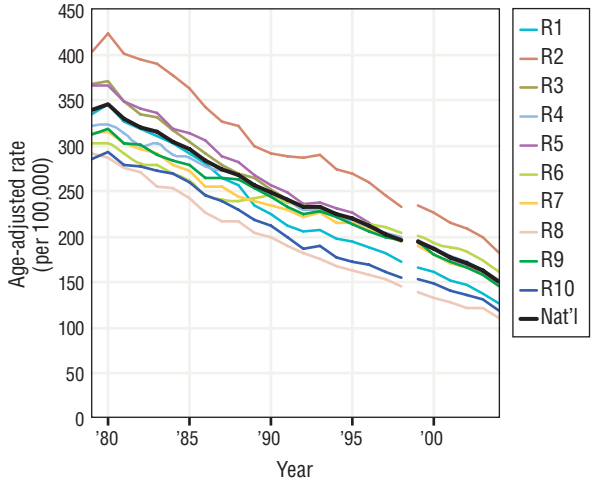
Among adults 18 years and older, the prevalence of heart disease and stroke between 1997 and 2006 has remained essentially the same (Exhibit 5-23). In contrast, the prevalence of hypertension has shown an increase from 191.6 cases per 1,000 in 1999 to 234.1 cases per 1,000 in 2006.

Gender, race, and age differences in CVD prevalence exist. The prevalence of coronary heart disease is consistently higher among males than among females (74.1 cases per 1,000 compared with 54.2 cases per 1,000 for women in 2006). In contrast, hypertension is more prevalent among women (238.4 cases per 1,000 for women compared with 229.5 for men in 2006). Among the racial groups reported, American Indians and Alaska Natives typically had the highest prevalence of coronary heart disease between 1999 and 2003. In 2006, however, whites had the highest prevalence of coronary heart disease (67.8 cases per 1,000), followed by American Indians and Alaska Natives (55.5 cases per 1,000), blacks or African Americans (52.0 cases per 1,000), and Asians (28.6 cases per 1,000). In 2006, Asians also consistently had the lowest prevalence of stroke (13.8 cases per 1,000) and hypertension (157.0 cases per 1,000) among the racial groups reported. In addition, the Hispanic or Latino population had a consistently lower prevalence of the major CVD-related diseases compared with the non-Hispanic or Latino population from 1999-2006, the period for which these data are available. For example, in 2006, prevalence in Hispanics or Latinos was lower than in non-Hispanics or Latinos for coronary heart disease (31.7 versus 68.6 cases per 1,000, respectively), hypertension (147.5 versus 247.0 cases per 1,000, respectively), and stroke (12.2 versus 27.6 cases per 1,000, respectively). (Data not shown.)

CVD Mortality

In 1998, the national age-adjusted CVD mortality rate (all types) was 352.0 per 100,000 compared to a rate of 541.0 per 100,000 in 1980 (Exhibit 5-24). This decline appears to continue after 1999, with the rate dropping from 349.3 per 100,000 in 1999 to 286.5 per 100,000 in 2004. Both coronary heart disease and stroke mortality rates have been declining in the U.S. The age-adjusted coronary heart disease mortality rate ranged from 345.2 per 100,000 in 1980 to 197.1 per 100,000 in 1998. For stroke mortality, the age-adjusted rate ranged from 97.1 per 100,000 in 1979 to 59.3 per 100,000 in 1998. The age-adjusted mortality rates for myocardial infarction ranged from 157.9 in 1979 to 76 per 100,000 in 1998. The age-adjusted mortality rates for coronary heart disease, stroke, and myocardial infarction in 2004 were 150.2, 50.0, and 52.3 per 100,000, respectively, compared to 194.6, 61.6, and 73.2 per 100,000, respectively, in 1999. Death rates from hypertension remained essentially the same between 1999 and 2004.

Exhibit 5-25. Age-adjusted coronary heart disease mortality rates in the U.S. by EPA Region, 1979-2004[a,b]



[a]Due to differences in the ICD system used for classifying mortality, data from 1979-1998 should not be directly compared to data from 1999-2004 [ICD-9 codes: 410-414, 429.2 (1979-1998); ICD-10 codes: I20-I25 (1999-2004)].

[b]Rates are age-adjusted to the 2000 U.S. standard population.

Data source: *CDC, 2007*

Both coronary heart disease and stroke mortality have been declining over time in each of the 10 EPA Regions (Exhibits 5-25 and 5-26). In 1979, coronary heart disease and stroke age-adjusted mortality rates ranged from 285.6 (Region 10) to 401.9 (Region 2) per 100,000 and 80.3 (Region 2) to 111.4 (Region 4) per 100,000, respectively. In 1998, coronary heart disease and stroke mortality rates ranged from 145.6 (Region 8) to 233.2 (Region 2) per 100,000 and 43.2 (Region 2) to 68.5 per (Region 10) 100,000, respectively. The observed decreases in coronary heart disease and stroke mortality also appear to continue in the 1999-2004 period.

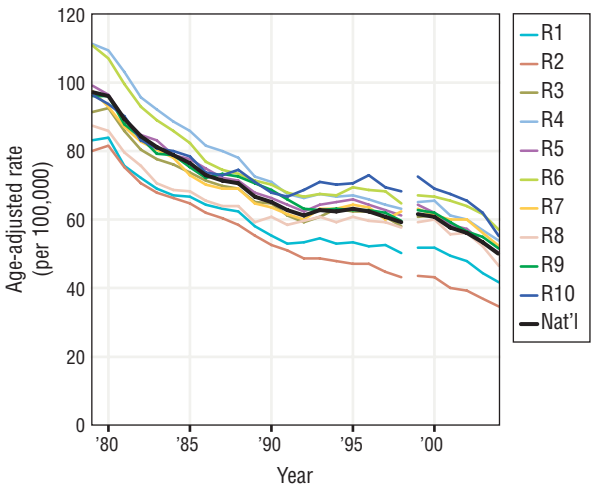
Differences exist in CVD mortality rates among gender, racial, and age groups. For example, in 2004, those age 65 and older had the highest CVD (all types), coronary heart disease, and stroke mortality (1,898.7, 990.8, and 346.2 per 100,000, respectively). For the same year, the age-adjusted CVD, coronary heart disease, and stroke mortality rates for

those 45 to 64 years of age were 172.7, 98.5, and 22.5 per 100,000, respectively. Notable differences in CVD (all types) and, specifically, coronary heart disease mortality rates exist between males and females, but not for stroke mortality. Coronary heart disease mortality among males in 2004 was 194.2 per 100,000, compared to 116.7 per 100,000 for women. In 2004, black or African American males had the highest CVD mortality rate at 451.1 per 100,000 compared to white males (333.6 per 100,000), black or African American females (331.0 per 100,000), and white females (236.7 per 100,000). (Data not shown.)

Indicator Limitations

- Prevalence data reported in the NHIS are based on self-reported responses to specific questions pertaining to CVD-related illnesses, and are subject to the biases associated with self-reported data. Self-reported data can underestimate the disease prevalence being measured if, for whatever reason, the respondent is not fully aware of his/her condition.
- All prevalence data are based on crude rates and are not age-adjusted, as CDC did not report age-adjusted data prior to 2002 in the data sources used for this indicator. Therefore, the reported disease prevalence rates across time or within different race and gender subgroups may not reflect differences in the age distribution of the populations being compared.
- For one or more years for which data are presented, coronary heart disease and stroke prevalence rates presented for Native Americans and Alaska Natives have a relative standard error of greater than 30 percent. In addition, stroke prevalence rates for one or more years for which data are presented for Asians have a relative standard error of greater than 30 percent. As such, these rates should be used with caution as they do not meet the standard of reliability or precision.
- CVD mortality rates are based on underlying cause of death as entered on a death certificate by a physician. Some individuals may have had competing causes of death. "When more than one cause or condition is entered by the physician, the underlying cause is determined by the sequence of conditions on the certificate, provisions of the ICD [International Classification of Diseases], and associated selection rules and modifications" (CDC, n.d.). Consequently, some misclassification of reported mortality might occur in individuals with competing causes of death, as well as the possible underreporting of CVD as the cause of death.
- The International Classification of Diseases 9th Revision (ICD-9) codes were used to specify underlying cause of death for years 1979-1998. Beginning in 1999, cause of death is specified with the International Classification of Diseases 10th Revision (ICD-10) codes. The two revisions differ substantially, and to prevent confusion about the significance of any specific disease code, data queries are separate.

Exhibit 5-26. Age-adjusted stroke mortality rates in the U.S. by EPA Region, 1979-2004[a,b]



[a]Due to differences in the ICD system used for classifying mortality, data from 1979-1998 should not be directly compared to data from 1999-2004 [ICD-9 codes: 430-434, 436-438 (1979-1998); ICD-10 codes: I60-I69 (1999-2004)].

[b]Rates are age-adjusted to the 2000 U.S. standard population.

Data source: *CDC, 2007*

Data Sources

CVD prevalence data were obtained from annual reports published by NCHS (NCHS, 1999-2007), which summarize health statistics compiled from the NHIS (http://www.cdc.gov/nchs/products/pubs/pubd/series/ser.htm). CVD mortality statistics were obtained from CDC's "compressed mortality" database, accessed through CDC WONDER (CDC, 2007) (http://wonder.cdc.gov/mortSQL.html). EPA Regional mortality statistics were generated by combining and age-adjusting state-by-state totals for each EPA Region using data from CDC WONDER.

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INDICATOR Chronic Obstructive Pulmonary Disease Prevalence and Mortality

Chronic obstructive pulmonary disease (COPD), sometimes referred to as chronic lung disease, is a disease that damages lung tissue or restricts airflow through the bronchioles and bronchi (NHLBI, 2003). Chronic bronchitis and emphysema are the most frequently occurring COPDs. Smoking is the most common cause of COPD, including cigarette, pipe, and cigar smoking (NHLBI, 2003). Other risk factors in the development and progression of COPD include asthma, exposure to air pollutants in the ambient air and workplace environment, genetic factors, and respiratory infections (CDC, 2003; American Lung Association, 2004).

Environmental tobacco smoke (ETS) may also increase the risk of developing COPD. The effect of chronic ETS exposure alone on pulmonary function in otherwise healthy adults is likely to be small. However, in combination with other exposures (e.g., prior smoking history, exposure to occupational irritants or ambient air pollutants), ETS exposure could contribute to chronic respiratory impairment. Children are especially sensitive to the respiratory effects of ETS exposure (State of California, 2005).

This indicator presents U.S. adult (age 18 and older) prevalence rates for chronic bronchitis and emphysema and mortality rates for COPD as a whole and for chronic bronchitis and emphysema. COPD prevalence data were compiled from 1999 to 2006 from the National Health Interview Survey (NHIS), conducted by the Centers for Disease Control and Prevention's (CDC's) National Center for Health Statistics (NCHS). The NHIS is the principal source of information on the health of the civilian non-institutionalized population of the U.S. and since 1960 has been one of the major data collection programs of NCHS. COPD prevalence is based on the number of adults who reported that they had ever been told by a doctor or other health practitioner that they had chronic bronchitis or emphysema. Mortality data (all ages) were compiled between 1979 and 2004 using the National Vital Statistics System (NVSS), maintained by NCHS. The NVSS registers virtually all deaths and births nationwide, with data coverage from 1933 to 2004 and from all 50 states and the District of Columbia.

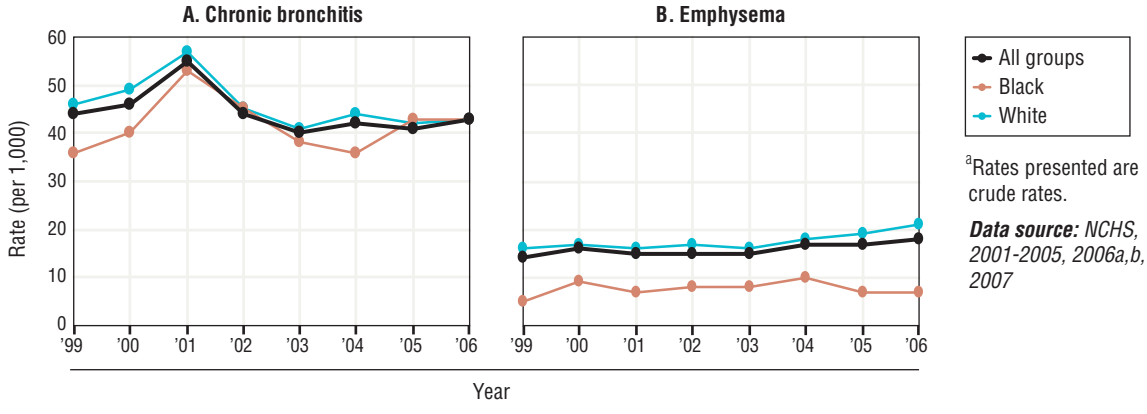
What the Data Show

COPD Prevalence

Exhibit 5-27 presents the prevalence of chronic bronchitis (panel A) and emphysema (panel B) from 1999 to 2006. The reported total prevalence of chronic bronchitis in U.S. adults over the age of 18 years ranged from a low of 40 (2003) to a high of 55 (2001) cases per 1,000. A small increase in prevalence of chronic bronchitis can be seen from 1999 to 2001, with a subsequent overall decline from 2001 to 2006. The reported total prevalence of emphysema in U.S. adults during the same time period ranged from 14 (1999) to 18 (2006) cases per 1,000. No notable change in the prevalence for emphysema was evident during this time period. Exhibit 5-27 also displays chronic bronchitis and emphysema prevalence by race. Chronic bronchitis prevalence was higher among white (designated as "white only") adults than black ("black or African American only") adults during 1999 (46 versus 36 cases per 1,000, respectively), 2000 (49 versus 40 cases per 1,000, respectively), and 2004 (44 versus 36 cases per 1,000, respectively). However, in 2006 rates in black and white adults are the same (43 cases per 1,000). Throughout the entire time period, emphysema prevalence is consistently higher among white adults than black adults.

In addition, the Hispanic or Latino population had a consistently lower prevalence of chronic bronchitis and

Exhibit 5-27. Chronic bronchitis and emphysema prevalence in U.S. adults (age 18 and older) by race, 1999-2006[a]



[a]Rates presented are crude rates.

Data source: *NCHS, 2001-2005, 2006a,b, 2007*

emphysema diseases than the non-Hispanic or Latino population from 1999-2006, the period for which these data are available. For example, in 2006, prevalence in Hispanics or Latinos was lower than non-Hispanics or Latinos for chronic bronchitis (22 compared to 46 cases per 1,000, respectively) and emphysema (4 compared to 21 cases per 1,000, respectively). (Data not shown.)

Gender differences are also seen. In 2006, females had about twice the reported prevalence of chronic bronchitis than males (57 versus 27 cases per 1,000 respectively), a consistently observed difference between 1997 and 2006. Unlike with chronic bronchitis, the prevalence rates for emphysema have been consistently higher in males than in females. (Data not shown.)

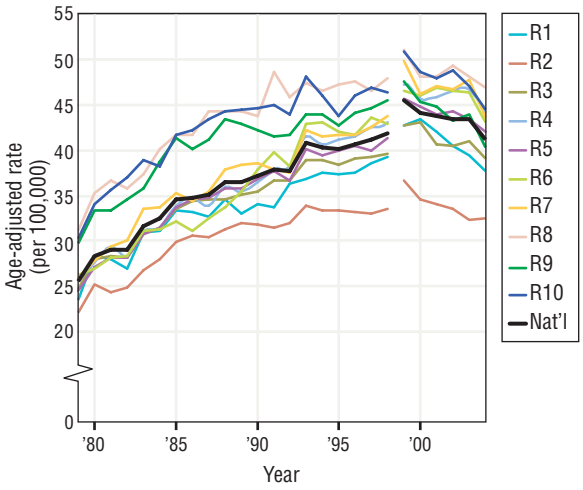
COPD Mortality

In 2004, COPD continues to be the fourth leading cause of mortality, accounting for 121,987 (5.1 percent) of all deaths (General Mortality indicator, p. 5-33). The age-adjusted mortality rate for COPD as a whole has increased over time, with rates ranging from 25.5 per 100,000 in 1979 to 41.8 per 100,000 in 1998. From 1999 to 2004, rates held steadier, ranging from 45.4 per 100,000 in 1999 to 41.1 per 100,000 in 2004. Mortality rates for emphysema (6.9 and 6.5 per 100,000 for 1979 and 1998, respectively, and 6.5 and 4.6 per 100,000 for 1999 and 2004, respectively) and chronic bronchitis (1.7 and 0.9 per 100,000 for 1979 and 1998, respectively, and 0.2 and 0.1 per 100,000 for 1999 and 2004, respectively) have not changed substantially during the same time period. (Data not shown.)

Exhibit 5-28 presents the overall COPD mortality rates in the U.S. and the 10 EPA Regions for 1979-1998 and 1999-2004. The age-adjusted COPD mortality rates have been increasing in each of the 10 Regions from 1979 to 1998. The rates ranged from 22.2 (Region 2) to 31.2 (Region 8) per 100,000 in 1979 and 33.5 (Region 2) to 47.9 (Region 8) per 100,000 in 1998. Between 1999 and 2004, COPD mortality rates in each of the 10 EPA Regions have generally declined.

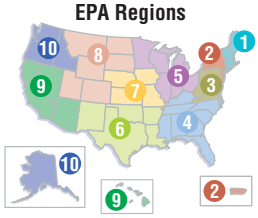
COPD age-adjusted mortality rates have been declining for males over time, with a rate of 58.7 per 100,000 in 1999 compared to 49.5 per 100,000 in 2004. For females, the rates are lower than males and have been relatively stable between 1999 and 2004 (37.7 and 36.0 per 100,000, respectively). The COPD age-adjusted mortality rate is higher among whites (43.2 per 100,000 in 2004) compared to blacks or African Americans (28.2 per 100,000 in 2004). COPD mortality rate increases with age: the 2004 rates were 0.3, 1.1, 21.0, and 284.3 per 100,000 for those age 0-14 years, 15-44 years, 45-64 years, and 65 years and older, respectively. (Data not shown.)

Exhibit 5-28. Age-adjusted chronic obstructive pulmonary disease mortality rates in the U.S. by EPA Region, 1979-2004[a,b]



[a]Due to differences in the ICD system used for classifying mortality, data from 1979-1998 should not be directly compared to data from 1999-2004 [ICD-9 codes: 490-494, 496 (1979-1998); ICD-10 codes: J40-J47 (1999-2004)].

[b]Rates are age-adjusted to the 2000 U.S. standard population.



Data source: *CDC, 2007*

Indicator Limitations

- Prevalence data presented in the NHIS are based on self-reported responses to specific questions pertaining to COPD-related illnesses, and are subject to the biases associated with self-reported data. Self-reported data can underestimate the disease prevalence being measured if, for whatever reason, the respondent is not fully aware of his/her condition.
- All prevalence data are based on crude rates and are not age-adjusted, as CDC did not report age-adjusted data prior to 2002 in the data sources used for this indicator. Therefore, the reported disease prevalence rates across time or within different race and gender subgroups may not reflect differences in the age distribution of the populations being compared.
- COPD mortality rates are based on underlying cause of death as entered on a death certificate by a physician. Some individuals may have had competing causes of death. "When more than one cause or condition is

INDICATOR Chronic Obstructive Pulmonary Disease Prevalence and Mortality *(continued)*

entered by the physician, the underlying cause is determined by the sequence of conditions on the certificate, provisions of the ICD [International Classification of Diseases], and associated selection rules and modifications" (CDC, n.d.). Consequently, some misclassification of reported mortality might occur in individuals with competing causes of death, as well as the possible underreporting of COPD as the cause of death.

- The International Classification of Diseases 9th Revision (ICD-9) codes were used to specify underlying cause of death for years 1979-1998. Beginning in 1999, cause of death is specified with the International Classification of Diseases 10th Revision (ICD-10) codes. The two revisions differ substantially, and to prevent confusion about the significance of any specific disease code, data queries are separate.

Data Sources

COPD prevalence data were obtained from annual reports published by NCHS (NCHS, 2001-2005, 2006a,b, 2007), which summarize health statistics compiled from the NHIS (http://www.cdc.gov/nchs/products/pubs/pubd/series/ser.htm). Mortality statistics were obtained from CDC's "compressed mortality" database, accessed through CDC WONDER (CDC, 2007) (http://wonder.cdc.gov/mortSQL.html). EPA Regional mortality statistics were generated by combining and age-adjusting state-by-state totals for each EPA Region using data from CDC WONDER.

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INDICATOR | Asthma Prevalence

Asthma is a chronic respiratory disease characterized by inflammation of the airways and lungs. During an asthma attack, the airways that carry air to the lungs are constricted, and as a result, less air is able to flow in and out of the lungs (NHLBI, 2004). Asthma attacks can cause a multitude of symptoms ranging in severity from mild to life-threatening. These symptoms include wheezing, breathlessness, chest tightness, and coughing (NHLBI, 2004). Currently, there is no cure for asthma; however, people who have asthma can still lead productive lives if they control their asthma. Taking medication and avoiding contact with environmental "triggers" can control asthma.

A family history of asthma contributes to susceptibility, but mostly what causes the development of asthma is unknown. Environmental exposures such as environmental tobacco smoke, dust mites, cockroach allergen, outdoor air pollution (e.g., ozone, particulate matter), pets, and mold are considered important triggers of an asthma attack (CDC, 2003, 2004; U.S. EPA, 2005, 2007).

Statistics for lifetime diagnosis prevalence, current asthma prevalence, and asthma attack prevalence are based on national estimates from the National Health Interview Survey (NHIS), conducted by the Centers for Disease Control and Prevention's (CDC's) National Center for Health Statistics (NCHS). The NHIS is the principal source of information on the health of the civilian non-institutionalized population of the U.S. and since 1960 has been one of the major data collection programs of NCHS. For this indicator, lifetime asthma diagnosis is defined as the number of adults/children who reported that they had ever been told by a doctor or other health practitioner that they had asthma. To determine current asthma prevalence, adults/children who had been told that they had asthma were asked whether they still have asthma. Asthma attack prevalence is based on the number of adults/children who reported an asthma episode or attack in the past 12 months.

What the Data Show

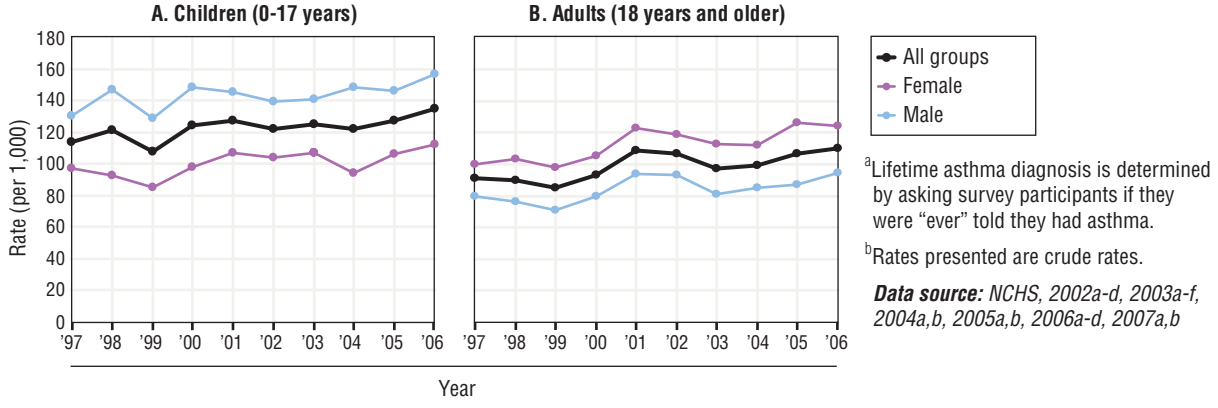
From 2003 to 2005, approximately 7.3 percent of the U.S. population reported that they currently have asthma (NCHS, 2007c). Reported asthma rates are highest in the child and adolescent population.

Adult Asthma

In adults, an increase in asthma prevalence rates (i.e., lifetime diagnosis) is evident from 1997 to 2001, with some decrease after 2001 and subsequent increase after 2003 (Exhibit 5-29, panel B). The prevalence rates range from a low of 85 cases per 1,000 in 1999 to a high of 110 cases per 1,000 in 2006. Asthma was consistently higher among adult females than males, with a range of 98 (1999) and 126 (2005) cases per 1,000 in females and 71 (1999) and 95 (2006) cases per 1,000 in males. The asthma prevalence rate also consistently decreases in older populations. In 2006, the asthma prevalence rates were 115 (ages 18-44 years), 105 (ages 45-64), 117 (ages 65-74 years), and 93 (ages 75+ years) cases per 1,000 (data not shown).

Exhibit 5-30 compares asthma rates across racial and ethnic groups for the 2003-2005 time period. As shown in panel A, the lifetime asthma diagnosis in adults was highest among American Indians/Alaska Natives (131 cases per 1,000), followed by blacks or African Americans (112 cases per 1,000), whites (100 cases per 1,000), and lowest among Asians (72 cases per 1,000). This same general pattern is seen for current asthma and asthma attack prevalence. Panel B shows that Hispanics or Latinos had lower rates across all three asthma prevalence categories than non-Hispanic whites and non-Hispanic blacks. For lifetime asthma diagnosis, 77 cases per 1,000 were reported in Hispanics or Latinos, 106 cases per 1,000 in non-Hispanic whites, and 111 cases per 1,000 in non-Hispanic blacks.

Exhibit 5-29. Estimated lifetime asthma diagnosis prevalence in children and adults in the U.S., 1997-2006[a,b]



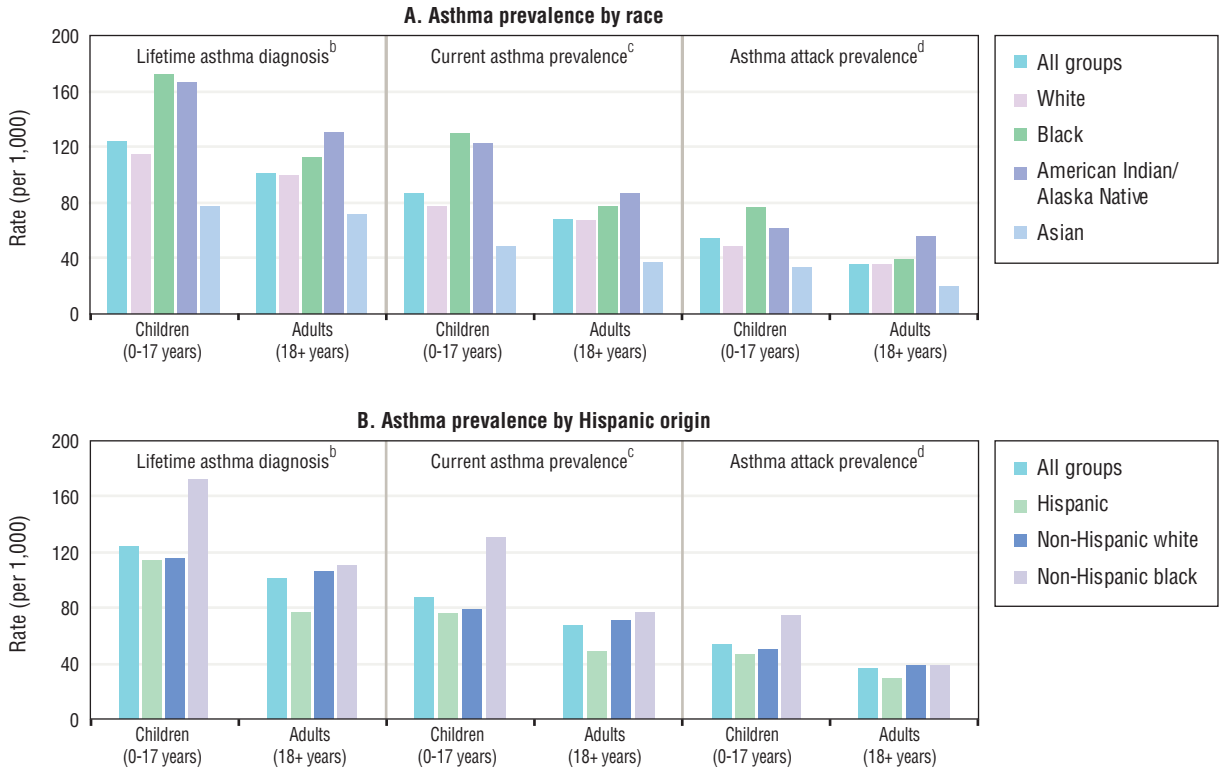
[a]Lifetime asthma diagnosis is determined by asking survey participants if they were "ever" told they had asthma.

[b]Rates presented are crude rates.

Data source: *NCHS, 2002a-d, 2003a-f, 2004a,b, 2005a,b, 2006a-d, 2007a,b*

INDICATOR | Asthma Prevalence *(continued)*

Exhibit 5-30. Asthma prevalence in the U.S. by race and Hispanic origin, 2003-2005[a]



[a]Rates presented for age 0-17 are crude rates; rates presented for age 18 and older are age-adjusted.

[b]Lifetime asthma diagnosis is determined by asking survey participants if they were "ever" told that they had asthma.

[c]Current asthma prevalence is determined by asking if the survey participant still has asthma.

[d]Asthma attack prevalence is determined by asking if the survey participant has had an asthma attack within the past 12 months.

Data source: *NCHS, 2007c*

Childhood Asthma

In 2006, almost 10 million children within the U.S. (age 0-17 years) were reported as ever having a diagnosis of asthma and nearly 4 million reported experiencing an asthma episode or attack during the previous 12 months. As shown in Exhibit 5-31, asthma prevalence rates increased approximately 4 percent per year between 1980 and 1996. Rates in subsequent years (1997-2006), reported in three categories, show no sharp upward or downward change through most of the time period, although an increase in current and lifetime reported asthma rates was observed in 2005 and 2006. Lifetime asthma diagnosis rates range from a low of 108 cases per 1,000 in 1999 to a high of 135 cases per 1,000 in 2006. Since tracking began in 2001, current asthma prevalence has ranged from approximately 83.4 cases per 1,000 (2002) to 93 cases per 1,000 (2006). Between 1997 and 2006, asthma attack prevalence rates have varied, with the lowest rate of 52.0 per 1,000 occurring in 2005 and the highest rate of 57.7 cases per 1,000 occurring in 2002. Male children consistently had higher rates of asthma prevalence than female children (Exhibit 5-29, panel A).

The overall pattern of asthma prevalence across races in children during 2003-2005 is similar to that seen in adults (Exhibit 5-30). One notable exception is that asthma prevalence in black or African American children was higher than asthma prevalence in American Indian/ Alaska Native children, the reverse of what was observed in the adult population. For example, reported lifetime asthma diagnosis was highest among black or African American children (172 cases per 1,000), followed by American Indians/Alaska Natives (166 cases per 1,000), whites (114 cases per 1,000), and Asians (78 cases per 1,000). Hispanic children had lower asthma prevalence rates for all three categories than non-Hispanic white and non-Hispanic black children.

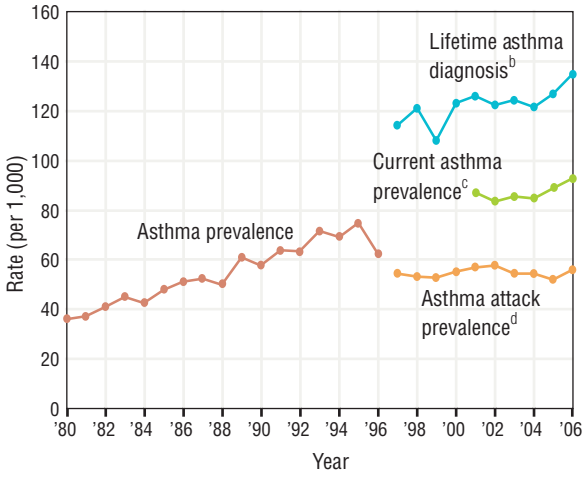
Indicator Limitations

- The NHIS questionnaire underwent major changes in 1997, and the data presented focus on surveys conducted from 1997 to the most currently available release (2004). The redesigned NHIS is different in content, format, and mode of data collection from earlier versions of the survey. Due to changes in methodology, comparisons between 1997–2004 NHIS estimates and pre-1997 NHIS data may not be valid.
- Prevalence data reported in the NHIS are based on self-reported responses to specific questions pertaining to airway-related illnesses, and are subject to the biases associated with self-reported data. Self-reported data may underestimate the disease prevalence being measured if, for whatever reason, the respondent is not fully aware of his/her condition.
- Except where otherwise noted, all prevalence data are based on crude rates and are not age-adjusted, as CDC did not report age-adjusted data prior to 2002 in the data sources used for this indicator. Therefore, the reported disease prevalence rates across time or within different race and gender subgroups may not reflect differences in the age distribution of the populations being compared.

Data Sources

Asthma prevalence data were obtained from annual reports published by NCHS (NCHS, 2002a–d; 2003a–f; 2004a,b; 2005a,b; 2006a–d; 2007a,b), which summarize health statistics compiled from the NHIS (http://www.cdc.gov/nchs/products/pubs/pubd/series/ser.htm#sr10). Race and ethnicity data were obtained from CDC's online "Health Data for All Ages" (NCHS, 2007c) (http://www.cdc.gov/nchs/health_data_for_all_ages.htm). The data used by CDC to create the asthma tables in "Health Data for All Ages" originate from the NHIS. The pre-1997 data also originate from the NHIS, as compiled by NCHS in Akinbami (2006).

Exhibit 5-31. Asthma prevalence in U.S. children (0-17 years), 1980-2006[a]



[a]Due to changes in NHIS questions in 1997, asthma prevalence data collected from 1980-1996 are not directly comparable to the data collected from 1997-2004.

[b]Lifetime asthma diagnosis is determined by asking survey participants if they were "ever" told their child has asthma.

[c]Current asthma prevalence is determined by asking if the child still has asthma.

[d]Asthma attack prevalence is determined by asking if the child has had an asthma attack within the past 12 months.

Data source: *Adapted from Akinbami, 2006; NCHS, 2007b*

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INDICATOR | Asthma Prevalence *(continued)*

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NCHS. 2003b. Summary health statistics for U.S. children: National Health Interview Survey, 2001. Vital Health Stat. 10(216). <http://www.cdc.gov/nchs/data/series/sr_10/sr10_216.pdf>

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NCHS. 2003e. Summary health statistics for U.S. adults: National Health Interview Survey, 1999. Vital Health Stat. 10(212). <http://www.cdc.gov/nchs/data/series/sr_10/sr10_212.pdf>

NCHS. 2003f. Summary health statistics for U.S. children: National Health Interview Survey, 1999. Vital Health Stat. 10(210). <http://www.cdc.gov/nchs/data/series/sr_10/sr10_210.pdf>

NCHS. 2002a. Summary health statistics for U.S. adults: National Health Interview Survey, 1998. Vital Health Stat. 10(209). <http://www.cdc.gov/nchs/data/series/sr_10/sr10_209.pdf>

NCHS. 2002b. Summary health statistics for U.S. children: National Health Interview Survey, 1998. Vital Health Stat. 10(208). <http://www.cdc.gov/nchs/data/series/sr_10/sr10_208.pdf>

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INDICATOR | Infectious Diseases Associated with Environmental Exposures or Conditions

Infectious diseases are human illnesses caused by viruses, bacteria, parasites, fungi, and other microbes. They can be spread by direct contact with an infected person or animal, through ingestion of contaminated food or water, by insects like mosquitoes or ticks (disease vectors), or by contact with contaminated surroundings like animal droppings or contaminated air. Demographic and environmental factors such as population growth, increased urbanization, and alteration of habitats of disease-carrying insects and animals (e.g., irrigation, deforestation) may promote the spread of infectious diseases (CDC, 1998a). The three broad infectious disease categories included here are those whose appearance and spread may be influenced to some extent by environmental conditions and change. They include gastrointestinal (GI) disease, arthropod-borne disease, and legionellosis.

- **Gastrointestinal diseases.** Eight notifiable GI diseases caused by microorganisms are discussed below: cholera, cryptosporidiosis, *Escherichia coli (E. coli)* O157:H7, giardiasis, hepatitis A, salmonellosis, shigellosis, and typhoid fever. The major environmental source of gastrointestinal illness is water or food that is contaminated with pathogenic microorganisms. The primary means of transmission for these eight diseases is through ingestion of contaminated food/water or through contact with and accidental ingestion of fecal matter (CDC, 2005a).
- **Arthropod-borne diseases.** Three arthropod-borne diseases are included: Lyme disease (transmission of *Borrelia burgdorferi* by ticks), Rocky Mountain spotted fever (transmission of *Rickettsia rickettsii* by ticks), and West Nile virus (transmitted by mosquitoes). Certain ticks and mosquitoes (arthropods) can carry bacteria and viruses that cause disease in humans. The arthropods acquire the bacteria or viruses when they bite an infected mammal or bird. Some studies indicate that spread of vector-borne disease may be influenced by land use and/or other environmental change (CDC, 2004). In recent years, both Lyme disease and West Nile virus have spread across the U.S. (CDC, 1993, 2000, 2004). Surveillance for Lyme disease was initiated by the Centers for Disease Control and Prevention (CDC) in 1982 (CDC, 1993).
- **Legionellosis.** Legionellosis, or Legionnaires' disease, is a serious and sometimes fatal form of pneumonia. It is caused by *Legionella* bacteria, which are found naturally in the environment and thrive in warm water and warm damp places. They are commonly found in lakes, rivers, creeks, hot springs, and other bodies of water. This bacterium has been associated with outbreaks in the U.S. linked to poorly maintained artificial water systems (e.g., air conditioning and industrial cooling systems) and air ventilation systems. Infection results from inhalation of contaminated water sprays or mists (CDC, 2003a).

This indicator reflects occurrence of these notifiable diseases as reported by health departments to the National Notifiable Diseases Surveillance System (NNDSS). A notifiable disease is one for which regular, frequent, and timely information regarding individual cases is considered necessary for the prevention and control of the disease (CDC, 2005b). Data are collected by all 50 states, five territories, New York City, and the District of Columbia, based on a list of recommended nationally notifiable infectious diseases, and compiled nationally. The temporal coverage of the data varies by disease. The number of states reporting may also vary. For example, in 1995, when cryptosporidiosis was first nationally reported, only 27 states reported; 45 states reported this disease by 1997.

What the Data Show

Gastrointestinal Diseases

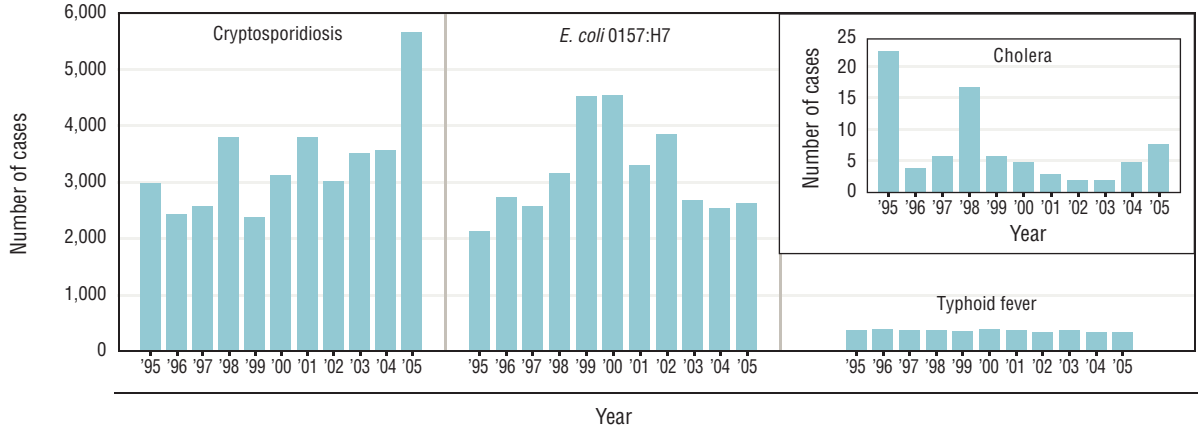
Exhibits 5-32 and 5-33 present the number of reported cases for each of the eight notifiable GI diseases from 1995-2005. In comparison to the other GI diseases, the number of newly identified cholera cases reported each year is low. From 1995 to 2005, just 81 laboratory-confirmed cases of cholera were reported to CDC, with eight cases being reported in 2005, the most current reporting year. Of these 81 total cases, 51 (63 percent) were acquired outside the U.S. The number of newly identified cases of typhoid fever was relatively stable from 1995 to 2005, ranging between a low of 321 cases in 2002 and a high of 396 cases in 1996. In 2005, 324 cases of typhoid fever were reported. Hepatitis A has continued to decline, with 31,582 cases reported in 1995 compared to 4,488 cases in 2005. The number of reported cryptosporidiosis cases increased in 2005 (5,659 cases). Fewer shigellosis cases were reported in 2004 and 2005 than in preceding years. No notable changes in the number of cases were observed for *E. coli* O157:H7, giardiasis (only 4 years of reporting data available), and salmonellosis.

Arthropod-Borne Diseases

Exhibit 5-34 presents the number of reported cases for three arthropod-borne diseases. Lyme disease is the most commonly reported arthropod-borne disease in the U.S., with 23,305 cases reported in 2005, just under the record number reported in 2002 (23,763 cases). CDC began surveillance of Rocky Mountain spotted fever in 1970. The number of new cases of Rocky Mountain spotted fever reported from 1995 to 2005 has fluctuated, ranging between a low of 365 cases in 1998 and a high of 1,936 cases in 2005. Cases of West Nile virus were first documented in the U.S. in 1999. A total of 80 cases were reported in 1999 (62 cases) and 2000 (18 cases) (data not shown). West Nile virus became nationally reportable in 2002, and the number of reported cases rose from 2,840 in

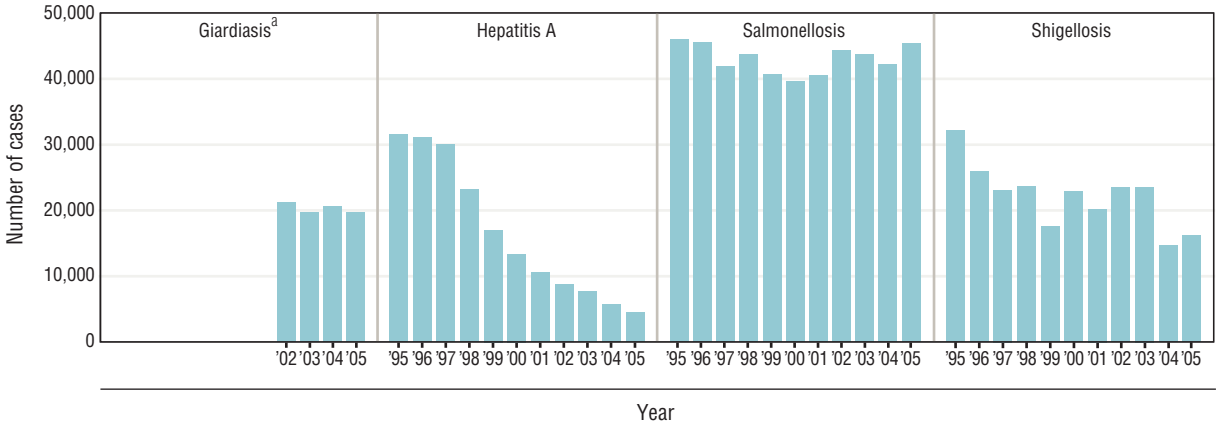
INDICATOR | Infectious Diseases Associated with Environmental Exposures or Conditions *(continued)*

Exhibit 5-32. Number of reported cases of gastrointestinal diseases in the U.S., 1995-2005 (part 1)



Data source: *CDC, 1996, 1997, 1998b, 1999, 2001, 2002, 2003b, 2004, 2005b, 2006, 2007*

Exhibit 5-33. Number of reported cases of gastrointestinal diseases in the U.S., 1995-2005 (part 2)



[a]Giardiasis was not on CDC's list of nationally notifiable infectious diseases prior to 2002.

Data source: *CDC, 1996, 1997, 1998b, 1999, 2001, 2002, 2003b, 2004, 2005b, 2006, 2007*

2002 to 2,866 in 2003. In 2004, the number of reported cases decreased to 1,142; the number increased to 1,309 reported cases in 2005.

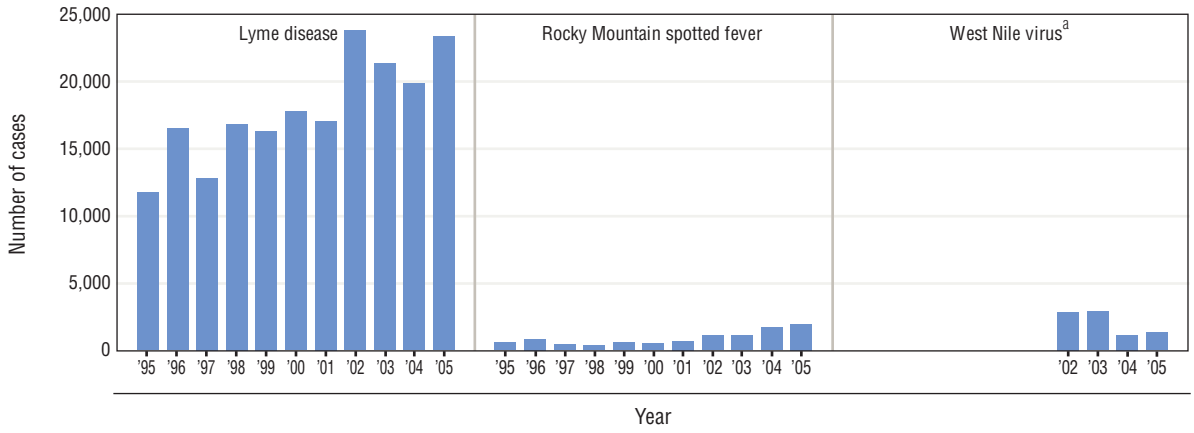
Legionellosis

Exhibit 5-35 presents the number of reported cases of legionellosis within the U.S. population from 1995 to 2005. From 1995 to 2002, the number of new cases of legionellosis was relatively stable, ranging from a low of 1,108 cases in 1999 to 1,355 cases in 1998. However, an increased number of new cases was reported in 2003 (2,232), 2004 (2,093), and 2005 (2,301).

Indicator Limitations

- State health departments report cases of notifiable diseases to CDC; policies for reporting can vary by disease or reporting jurisdiction.
- Disease reporting likely underestimates the actual number of cases for a given time period because reporting nationally notifiable diseases to CDC is voluntary. Additionally, the completeness of reporting likely varies by disease. The degree of completeness of data reporting is influenced by many factors such as the diagnostic facilities available, the control measures in effect, public awareness of a specific

Exhibit 5-34. Number of reported cases of arthropod-borne diseases in the U.S., 1995-2005



[a]West Nile virus was not on CDC's list of nationally notifiable infectious diseases prior to 2002.

Data source: *CDC, 1996, 1997, 1998b, 1999, 2001, 2002, 2003b, 2004, 2005b, 2006, 2007*

disease, and the interests, resources, and priorities of state and local officials responsible for disease control and public health surveillance (CDC, 2007).

- Factors such as changes in case definitions for public health surveillance, introduction of new diagnostic tests, or discovery of new disease entities can cause changes in disease reporting that are independent of the true incidence of disease (CDC, 2004).
- Prior to 2005, only confirmed "neuroinvasive" cases of West Nile virus—the most severe form of the condition—were reported (CDC, 2005c). Beginning in 2005, non-neuroinvasive domestic arboviral diseases for the six domestic arboviruses listed were added to the list of nationally notifiable diseases; these included West Nile fever, a non-neuroinvasive form of West Nile virus (CDC, 2007). In order to maintain reporting consistency, only neuroinvasive cases are presented for this indicator.

Data Sources

The data for this indicator were obtained from CDC annual reports that summarize data on nationally notifiable infectious diseases reported to CDC by state health agencies across the country (CDC, 1996, 1997, 1998b, 1999, 2001, 2002, 2003b, 2004, 2005b, 2006, 2007). Data are collected and compiled from reports sent by state health departments to the NNDSS, which is operated by CDC. The NNDSS is neither a single surveillance system nor a method of reporting. Certain NNDSS data are reported to CDC through separate surveillance information systems and through different reporting mechanisms; however, these data are aggregated and compiled for publication purposes (CDC, 2007).

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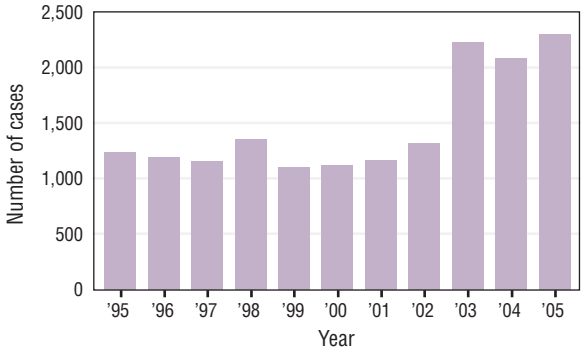
Exhibit 5-35. Number of reported cases of legionellosis in the U.S., 1995-2005



Data source: *CDC, 1996, 1997, 1998b, 1999, 2001, 2002, 2003b, 2004, 2005b, 2006, 2007*

INDICATOR | Infectious Diseases Associated with Environmental Exposures or Conditions *(continued)*

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INDICATOR | Birth Defects Prevalence and Mortality

Birth defects are structural or functional anomalies causing physical or mental disability, some of which can be fatal. Although birth defects are the leading cause of infant mortality (deaths occurring to those under 1 year of age) in the U.S., the cause is unknown for approximately 70 percent of all cases (Infant Mortality indicator, p. 5-36) (CDC, 2005). Many different factors may be associated with the development of birth defects, such as genetic and/or chromosomal aberrations, *in utero* exposure to viruses or bacteria, uncontrolled maternal diabetes, maternal cigarette smoke, maternal use of drugs and alcohol during pregnancy, and prenatal exposure to chemicals. All of these factors may influence normal infant growth or development, resulting in different types of birth defects (NICHD, 2006).

This indicator presents birth defects prevalence at birth and mortality rates among infants in the U.S. as recorded in the National Vital Statistics System, which registers virtually all births and deaths nationwide. Data collection began in 1933 and is available through 2004. Birth defects data are collected on death certificates from all 50 states and the District of Columbia and recorded on birth certificates for 49 states and the District of Columbia. Reported race and ethnicity data are based on the race and ethnicity of the mother.

What the Data Show

Exhibit 5-36 presents the prevalence of live births with identified specific congenital anomalies (i.e., birth defects) between 1999 and 2004. The most frequently occurring

Exhibit 5-36. Prevalence of live births in the U.S. with specific birth defects (congenital anomalies), 1999-2004[a]

	1999	2000	2001	2002	2003	2004
Overall rate	1,170.2	1,164.2	1,178.8	1,170.6	1,103.4	1,111.8
Central nervous system anomalies						
Anencephalus	11.0	10.7	9.9	9.9	11.4	10.9
Spina bifida/meningocele	20.1	20.7	19.9	20.0	18.7	19.3
Hydrocephalus	21.5	23.7	22.5	22.5	22.2	22.4
Microcephalus	5.9	7.2	5.6	5.5	5.6	6.9
Other central nervous system anomalies	20.0	20.7	24.8	22.2	21.1	21.5
Circulatory/respiratory anomalies						
Heart malformations	119.8	124.9	122.5	129.9	128.9	137.7
Other circulatory/respiratory anomalies	140.6	138.1	139.6	131.7	126.1	135.3
Gastrointestinal anomalies						
Rectal atresia/stenosis	9.0	8.4	9.0	8.3	7.8	8.7
Tracheo-esophageal fistula/esophageal atresia	13.3	12.1	12.0	10.8	10.8	11.8
Omphalocele/gastroschisis	30.2	29.7	31.8	30.3	32.5	31.9
Other gastrointestinal anomalies	29.8	29.9	34.2	36.1	33.0	33.9
Urogenital anomalies						
Malformed genitalia	76.3	84.2	88.4	86.6	79.7	80.8
Renal agenesis	13.7	13.8	14.8	15.4	14.0	13.6
Other urogenital anomalies	99.0	99.3	102.8	101.8	90.2	89.5
Chromosomal anomalies						
Cleft lip/palate	80.9	82.1	80.6	78.5	75.9	77.7
Polydactyly/syndactyly/adactyly	87.9	87.2	82.4	82.2	76.4	74.8
Clubfoot	55.7	57.2	58.6	59.6	57.6	55.7
Diaphragmatic hernia	13.1	10.8	11.4	12.1	11.4	10.4
Other musculoskeletal/integumental anomalies	239.9	217.0	226.4	228.9	208.2	211.1
Down's syndrome	45.5	46.9	45.5	46.7	46.5	47.9
Other chromosomal anomalies	36.9	39.7	36.2	31.6	30.1	29.3

[a]Rates are per 100,000 live births.

Data source: *NCHS, 2001, 2002a,b, 2003, 2005, 2006; CDC, 2007a*

types of birth defects were various musculoskeletal/integumental anomalies, circulatory/respiratory system anomalies, and heart malformations. In 2004, heart malformations occurred at a rate of 137.7 per 100,000 live births, which was highest among the specific anomalies listed (i.e., categories that do not include "other"). The overall rate of birth defects (i.e., all birth defects combined) has been relatively stable between 1999 and 2002, with a noticeable decline in 2003 and 2004. Blacks have a consistently higher rate of birth defects than whites during this time period, with a rate of 1,337.5 (blacks) compared with 1,064.0 (whites) birth defects per 100,000 live births in 2004 (data not shown).

Rates for certain types of anomalies differ widely with maternal age. For example, in 2004 as in past years, infants of the youngest mothers (under 20 years of age) have the highest rates for omphalocele/gastroschisis, a defect or abnormality of the anterior abdominal wall (87.1 per 1,000 live births); infants of mothers age 35 years and over have the highest rates for Down's syndrome (348.3 per 1,000 live births). (Data not shown.)

INDICATOR | Birth Defects Prevalence and Mortality *(continued)*

Birth defects continue to be the leading cause of infant mortality, accounting for 5,622 (20.1 percent) of the 27,936 infant deaths in 2004 (Exhibit 5-16, Infant Mortality indicator, p. 5-37). Between 1979 and 1998, a decline in the national birth defects mortality rate has been observed, ranging from 255.4 per 100,000 live births in 1979 to 157.6 per 100,000 live births in 1998. From 1999 to 2004, the birth defects mortality rates were 144.2 (1999), 150.9 (2000), 136.7 (2001), 139.4 (2002), 140.4 (2003), and 137.9 (2004) per 100,000 live births. (Data not shown.)

Birth defects mortality was consistently higher among black infants than white infants. In 2004, for example, mortality attributed to birth defects among black male and female infants was 169.9 and 155.6 per 100,000 infants, respectively; among white male and female infants, it was 134.3 and 134.7 per 100,000 infants, respectively. (Data not shown.)

Indicator Limitations

- Because some birth defects are not recognized immediately, they are often underreported on both birth and death certificates (Friis and Sellers, 1999). Many anomalies are hard to detect at birth, which limits early ascertainment and complete reporting. The most serious and/or apparent anomalies are more likely to be identified and reported prior to hospital discharge (Honein et al., 2001).
- The lack of uniform reporting on birth certificates introduces additional uncertainty. For example, race information may be missing or incomplete. Also, beginning in 2003, two states began using a revised "standard certificate of live birth;" therefore, a subset of anomaly data was excluded because of the lack of comparability with other data sets (NCHS, 2005).
- The congenital anomalies reported on birth certificates are rare events. Since a small change in the number of anomalies reported can result in a relatively large change in rates, caution should also be used in comparing yearly rates for a specific anomaly.
- The birth defects anomaly groupings that include "other" (e.g., other musculoskeletal anomalies) include a large number of non-specific birth defects and should be considered separately from the specific birth defects listed.
- Birth defects mortality rates are based on underlying cause of death as entered on a death certificate by a physician. Incorrect coding and low rates of autopsies that confirm the cause of death may occur. Additionally, some individuals may have had competing causes of death. "When more than one cause or condition is entered by the physician, the underlying cause is determined by the sequence of conditions on the certificate, provisions of the ICD [International Classification of Diseases], and associated selection rules and modifications" (CDC, n.d.). Consequently, some misclassification of reported mortality might occur in individuals with competing causes of death, as well as underreporting of some birth defects as the cause of death.
- The International Classification of Diseases 9th Revision (ICD-9) codes were used to specify underlying cause of death for years 1979-1998. Beginning in 1999, cause of death is specified with the International Classification of Diseases 10th Revision (ICD-10) codes. The two revisions differ substantially, and to prevent confusion about the significance of any specific disease code, data queries are separate. The relatively large difference between birth defects mortality rates reported from 1979 through 1998 and those reported beginning in 1999 may be due to some changes in the criteria used to report birth defects mortality during the switch from ICD-9 to ICD-10.

Data Sources

The birth defects rate data used for this indicator are from National Vital Statistics Reports published by the CDC's National Center for Health Statistics (NCHS, 2001, 2002a,b, 2003, 2005, 2006). CDC's "VitalStats"—a collection of vital statistics products including tables, data files, and reports that allow users to access and examine vital statistics and population data interactively—were used to obtain specific anomaly data for 2004 (CDC, 2007a). The birth defects mortality data were obtained from a published report by the National Center for Health Statistics (NCHS, 2007) and from CDC's compressed mortality files (underlying cause of death), accessed via CDC WONDER (CDC, 2007b), at http://wonder.cdc.gov.

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CDC. 2005. Birth defects. Accessed September 2007. <http://www.cdc.gov/ncbddd/bd/facts.htm>

CDC. n.d. CDC WONDER: Help page for compressed mortality file. Accessed September 2007. <http://wonder.cdc.gov/wonder/help/cmf.html>

Friis, R.H., and T.A. Sellers. 1999. Epidemiology for public health practice. Second edition. Gaithersburg, MD: Aspen Publishers.

INDICATOR | Birth Defects Prevalence and Mortality *(continued)*

Honein, M.A., L.J. Paulozzi, and M.L. Watkins. 2001. Maternal smoking and birth defects: Validity of birth data for effect estimation. Public Health Reports 116:327-335

NCHS (National Center for Health Statistics). 2007. Deaths: Final data for 2004. National Vital Statistics Reports 55(19). <http://www.cdc.gov/nchs/data/nvsr/nvsr55/nvsr55_19.pdf>

NCHS. 2006. Births: Final data for 2004. National Vital Statistics Reports 55(1). <http://www.cdc.gov/nchs/data/nvsr/nvsr55/nvsr55_01.pdf> See Table 25.

NCHS. 2005. Births: Final data for 2003. National Vital Statistics Reports 54(2). <http://www.cdc.gov/nchs/data/nvsr/nvsr54/nvsr54_02.pdf> See Table 49.

NCHS. 2003. Births: Final data for 2002. National Vital Statistics Reports 52(10). <http://www.cdc.gov/nchs/data/nvsr/nvsr52/nvsr52_10.pdf> See Table 49.

NCHS. 2002a. Births: Final data for 2001. National Vital Statistics Reports 51(2). <http://www.cdc.gov/nchs/data/nvsr/nvsr51/nvsr51_02.pdf> See Table 49.

NCHS. 2002b. Births: Final data for 2000. National Vital Statistics Reports 50(5). <http://www.cdc.gov/nchs/data/nvsr/nvsr50/nvsr50_05.pdf> See Table 49.

NCHS. 2001. Births: Final data for 1999. National Vital Statistics Reports 49(1). <http://www.cdc.gov/nchs/data/nvsr/nvsr49/nvsr49_01.pdf> See Table 49.

NICHD (National Institute of Child Health and Human Development). 2006. Birth defects and developmental disabilities. Accessed September 2007. <http://www.nichd.nih.gov/womenshealth/research/pregbirth/birthdefects.cfm>

INDICATOR | Low Birthweight

The term "low birthweight" (LBW) is typically used for any infant weighing less than 2,500 grams at birth. Weight is a critical health measure because LBW children are more prone to death and disability than their counterparts.

The etiology of LBW for term-LBW (born after 37+ weeks of gestation) infants and preterm-LBW (born after less than 37 weeks of gestation) infants differs. For term-LBW infants, underlying causes include factors such as maternal smoking, weight at conception, and gestational weight gain, whereas for preterm-LBW infants, the etiology largely remains unexplained (CDC, 1994). Various exposures have been implicated as risk factors for term-LBW (e.g., maternal smoking, maternal exposure to lead, diethylstilbestrol, occupational exposures) (Sram et al., 2005; Kiely et al., 1994). The potential effect of air pollution on LBW continues to be researched (e.g., particulate matter, carbon monoxide, ozone).

This indicator presents the percentage of LBW infants born in the U.S. based on natality data reported to the National Vital Statistics System (NVSS). The NVSS registers virtually all deaths and births nationwide, with data coverage from 1933 to 2004 and from all 50 states and the District of Columbia.

The data presented are based on singleton births only. This was done to eliminate the effect of multiple births. The data are presented across three maternal age groups (under 20 years, 20-39 years, and 40 years and older). Additionally, the data are stratified and reported for preterm (less than 37 weeks) and full-term (37 weeks and over) births because of the strong association between birthweight and gestational age.

What the Data Show

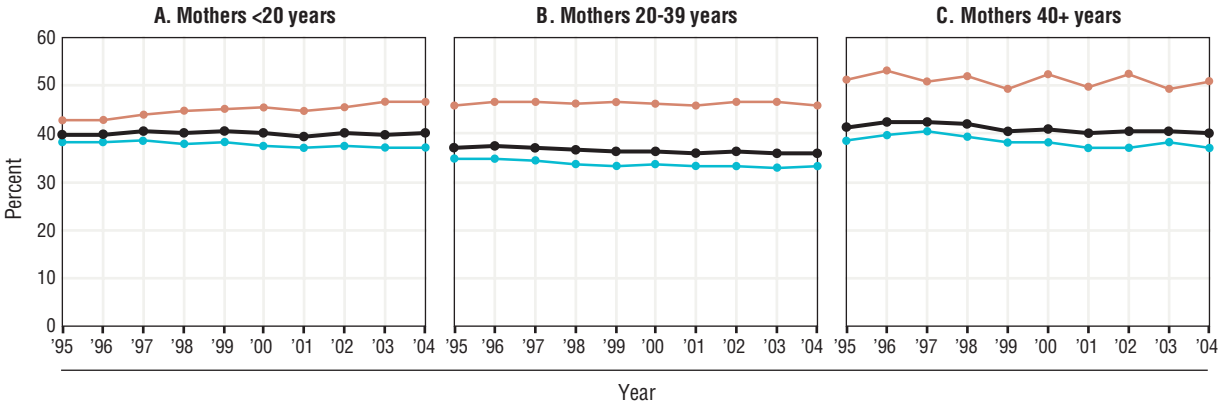
As expected, the percent of total LBW deliveries among preterm births is much higher than the percent of total LBW deliveries among full-term births across each of the three maternal age categories (Exhibits 5-37 and 5-38).

In general, small differences in the percent of LBW babies among maternal age categories are evident for both pre- and full-term births. For example, in 2004, the frequency of LBW babies among full-term births for mothers less than 20 years old (4.0 percent) is almost 1 percent higher than for mothers who are 40 years and older (3.2 percent) and about 1.4 percent higher than for mothers who are in the 20-39 age group (2.4 percent) (Exhibit 5-38).

Among the full-term births, black women had consistently higher frequencies of LBW babies compared to any of the other racial groups reported from 1995 and 2004. This racial pattern is evident in 2004 for all three maternal age groups, and the difference is most apparent in the 40 and older age group (6.2 percent for blacks and 2.7 percent for whites) (Exhibit 5-38).

The percentages of term-LBW babies among the other two racial groups reported in 2004, Native Americans and Asians/Pacific Islanders, were 4.1 percent and 3.3 percent, respectively, for the 40 and older age group. In 2004, some

Exhibit 5-37. Percent of low birthweight infants (<2,500 grams) born preterm in the U.S. by mother's race and age, 1995-2004[a,b]

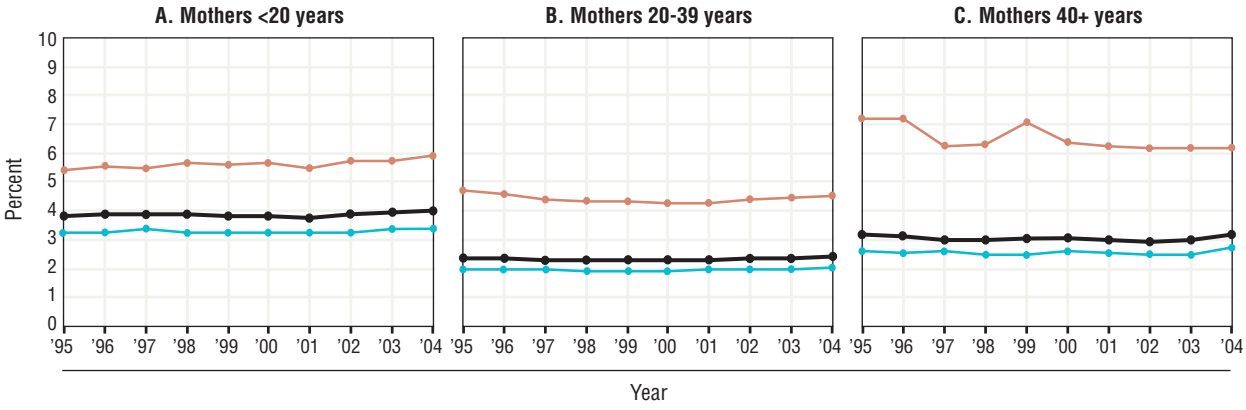


[a]Preterm births are births occurring at <37 weeks gestation.

[b]Data represent singleton births only.

Data source: *CDC, 2007*

Exhibit 5-38. Percent of low birthweight infants (<2,500 grams) born full-term in the U.S. by mother's race and age, 1995-2004[a,b]



[a]Full-term births are births occurring at ≥ 37 weeks gestation.

[b]Data represent singleton births only.

Data source: *CDC, 2007*

variation in the frequency of term-LBW was reported for Native Americans and Asian/Pacific Islanders among the three different age groups reported (under 20 years, 20-39 years, and 40 years and older), with Asian/Pacific Islanders showing the highest percentage of LBW babies (4.7 percent) among the under 20 year age group and Native Americans showing the highest percentage of LBW babies (4.1 percent) among women 40 years and older. Hispanic women and non-Hispanic women had similar frequencies of LBW babies. For example, in 2004, the percent of LBW babies for Hispanic women was 2.4 percent compared to 2.7 percent for non-Hispanic women. (Data not shown.)

Indicator Limitations

- Complete reporting of natality indicators such as LBW may vary due to differences in the reporting requirements established by each state. In some states, the number of LBW babies may be underreported.

INDICATOR | Low Birthweight *(continued)*

Data Source

The data used for this indicator were public-use natality data (1995-2002 and 2003-2004) obtained from the Centers for Disease Control and Prevention's National Center for Health Statistics, Division of Vital Statistics, available via CDC WONDER (CDC, 2007), at http://wonder.cdc.gov.

References

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Kiely, J.S., K.M. Brett, S. Yu, and D.L. Rowley. 1994. Low birthweight and intrauterine growth retardation. In: Wilcox, L.S., and J.S. Marks, eds. From data to action: CDC's public health surveillance for women, infants, and children. CDC's maternal and child health monograph 1994. Atlanta, GA: Centers for Disease Control and Prevention.

Sram R.J., B. Binkova, J. Dejmek, and M. Bobak. 2005. Ambient air pollution and pregnancy outcomes: A review of the literature. Environ. Health Perspect. 113(4):375-382.

INDICATOR | Preterm Delivery

Preterm delivery is defined as delivery prior to 37 weeks of gestation (a typical pregnancy lasts 40 weeks). The shorter the gestational age of an infant, the more likely (s)he is to suffer adverse effects. Preterm birth along with low birthweight is the second leading cause of infant death (Infant Mortality indicator, p. 5-36) (NCHS, 2004, 2006), and accounts for nearly half of all congenital neurological defects, such as cerebral palsy, and more than two-thirds of infant deaths (Goldenberg and Rouse, 1998; NCHS, 2006).

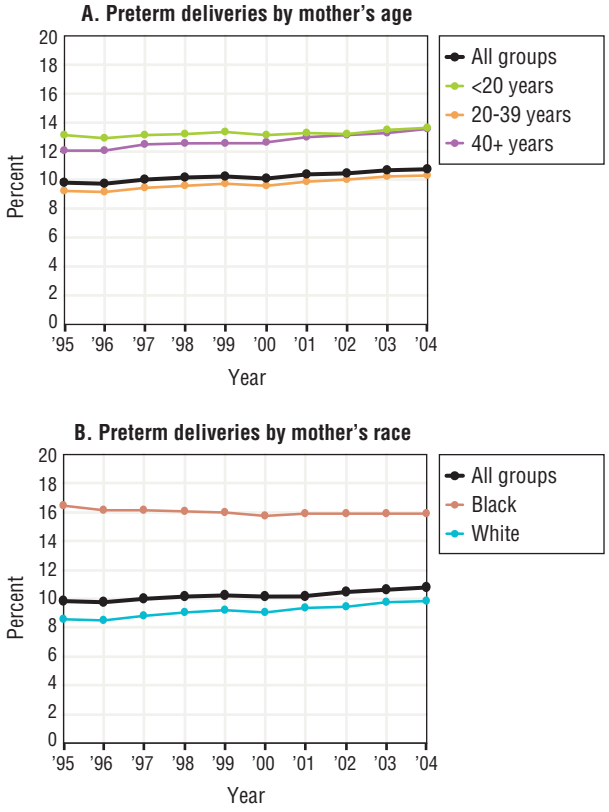
The determinants of preterm births are not fully known and the causes are often multi-factorial. Maternal high-risk conditions (e.g., infertility problems, vaginal spotting, inadequate maternal weight gain), previous history, socioeconomic status, smoking, alcohol consumption before third trimester, and multiple gestation pregnancy are known risk factors for preterm delivery. Environmental contaminants (e.g., lead, environmental tobacco smoke, air pollution) continue to be studied to better understand the strength of the associations with preterm delivery.

This indicator presents the proportion of U.S. infants born prior to 37 weeks of gestation, based on natality data reported to the National Vital Statistics System (NVSS). The NVSS registers virtually all deaths and births nationwide, with data coverage from 1933 to 2004 and from all 50 states and the District of Columbia. The data presented here on preterm delivery were based on singleton births only. This was done to eliminate the effect of multiple births. The data are presented across three maternal age groups (under 20 years, 20-39 years, and 40 years and older).

What the Data Show

The proportion of infants defined as preterm has risen 18 percent since 1990 (NCHS, 2006). A small overall increase in preterm births has been observed from 1995 (9.8 percent)

Exhibit 5-39. Preterm deliveries in the U.S. by mother's age and race, 1995-2004[a,b]



[a]Preterm deliveries are births occurring at <37 weeks gestation.

[b]Data represent singleton births only.

Data source: *CDC, 2007*

INDICATOR | Preterm Delivery *(continued)*

to 2004 (10.8 percent). The largest percent increase between 1995 and 2000 has occurred among mothers in the 40 and over age group, with the percent of preterm births ranging from 12.0 (1995) to 13.5 percent (2004). The next largest percent increase was observed in the 20–39 year old maternal group, ranging from 9.2 percent (1996) to 10.3 percent (2004), with little overall change over time among those under 20 years of age (Exhibit 5-39, panel A).

In 1995, the percent of preterm births was almost twice as high among black mothers as among white mothers (16.4 versus 8.5 percent) (Exhibit 5-39, panel B). From 1995 to 2004, preterm delivery among black mothers decreased slightly: from 16.4 percent in 1995 to 15.9 percent in 2001, where the percentage has remained the same through 2004. During the same time, preterm delivery among white mothers increased slightly, rising from 8.5 percent in 1995 to 9.9 percent in 2004, resulting in a slight narrowing of the difference in the preterm birth rate between black and white mothers. Preterm delivery for Hispanic mothers ranged from 10.1 (1995) to 10.9 percent (2004), compared to 9.7 (1996) and 10.7 (2004) percent for non-Hispanic mothers between 1995 and 2004. (Data not shown.)

Indicator Limitations

- The primary measure used to determine the gestational age of the newborn is the interval between the first day of the mother's last normal menstrual period (LMP) and the date of birth. This measurement is subject to error for reasons such as imperfect maternal recall or misidentification of the LMP because of postconception bleeding, delayed ovulation, or intervening early miscarriage. When the LMP and date of birth are clearly inconsistent with the infant's birthweight or plurality, then a "clinical estimate of gestation" is used. Problems with reporting gestational age persist and may occur more frequently among some subpopulations and among births with shorter gestations (NCHS, 2006).

Data Source

The data used for this indicator were public-use natality data (1995–2002 and 2003–2004) obtained from the Centers for Disease Control and Prevention's National Center for Health Statistics, Division of Vital Statistics, available via CDC WONDER (CDC, 2007), at http://wonder.cdc.gov.

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NCHS (National Center for Health Statistics). 2006. Births: Final data for 2004. National Vital Statistics Reports 55(1). <http://www.cdc.gov/nchs/data/nvsr/nvsr55/nvsr55_01.pdf>

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5.4.3 Discussion

What These Indicators Say About Trends in Human Disease and Conditions for Which Environmental Contaminants May Be a Risk Factor

The indicators selected to answer this question represent diseases and conditions that affect multiple systems of the human body and are associated with a number of risk factors, some of which include exposures to contaminants that may be found in the air, water, and land. Some indicators represent chronic conditions (e.g., various cancers, heart and lung disease), some are primarily acute in nature (e.g., infectious diseases), and others represent conditions of the developing fetus and neonate. Understandably, no striking trends are evident across the broad categories of diseases represented by the indicators. However, some changes in disease rates or occurrence were observed for individual indicators. These relate largely to disease patterns observed over time and to differences observed across age groups, gender, and racial and ethnic groups.

Generally, the occurrence of many chronic diseases in adults is increasing with the aging of the population (Cancer indicator, p. 5-43; Cardiovascular Disease indicator, p. 5-48; Chronic Obstructive Pulmonary Disease indicator, p. 5-52). However, while overall cancer incidence rates showed a steady increase from the mid-1970s to the mid-1990s, rates have held relatively steady between 1997 and 2004. With the exception of prostate cancer in males and breast cancer in females, site-specific cancer rates also have remained fairly constant. Similarly, prevalence rates for cardiovascular disease and chronic obstructive pulmonary disease have shown no striking changes between 1997 and 2006, with the exception of an overall increase in the prevalence of hypertension during this time period. Prevalence rates for adult asthma have fluctuated from 1997 to 2006, with an overall increase during that time period (Asthma indicator, p. 5-55).

No distinct upward or downward patterns were revealed between 1995 and 2005 for most of the acute infectious gastrointestinal diseases presented in this report. An exception is the decrease in hepatitis A cases, which has been attributed to childhood vaccination for this disease.[57] Other observable shifts in acute infectious diseases, such as an increase of cryptosporidiosis in 2005, are difficult to interpret because of acknowledged uncertainties in the completeness of disease reporting in a given year.[58] Generally increased reported occurrence of arthropod-borne diseases and legionellosis bears watching (Infectious Diseases indicator, p. 5-59).

Review of diseases in children and birth outcomes revealed the following overall trends. Childhood cancer incidence has increased slightly since 1975, with boys having a higher incidence rate than girls. Leukemia and brain and other nervous system cancers remain the leading cancer sites in children (Childhood Cancer indicator, p. 5-46). Prevalence rates for childhood asthma remain at historically high levels following increases from 1980 through the late 1990s (Asthma indicator, p. 5-55).[59] A wide range of birth defects continues to be reported each year, but with no notable shifts in prevalence observed for specific types of defects from 1999 to 2004. Heart malformations and other circulatory/respiratory anomalies and musculoskeletal/integumental anomalies remain the most prevalent types of birth defects based on birth certificate data (Birth Defects indicator, p. 5-62). Among full-term singleton births, the percentage of low birthweight infants has not varied from 1995 to 2004. Age of mother showed the greatest influence, with the greatest number of low birthweight infants born to younger mothers (less than 20 years old) (Low Birthweight indicator, p. 5-65). The highest rate of preterm births is also seen in these younger mothers, though nearly comparable and rising preterm birth rates are seen among mothers over the age of 40 (Preterm Delivery indicator, p. 5-67).

Some differences were observed across racial and ethnic groups. Observations are reported for the most recently available annual data set. Overall, cancer incidence is higher among black males than for any other racial group. Less disparity was observed between cancer incidence in white and black women. With childhood cancers, higher rates have been consistently reported in whites than in blacks (Cancer indicator, p. 5-43, Childhood Cancer indicator, p. 5-46). For cardiovascular disease (p. 5-48), prevalence rates were generally reported highest among whites and American Indians/Alaska Natives, followed by blacks or African Americans and Asians. Asthma rates were generally reported highest among blacks or African Americans in children and American Indians/Alaska Natives in adults, followed by whites and Asians (Asthma indicator, p. 5-55).

The percentage of preterm and low birthweight infants is consistently higher among blacks than whites (1.5 to nearly 3 times higher). This observation is seen across all maternal age groups (Preterm Delivery indicator, p. 5-67; Low Birthweight indicator, p. 5-65). When available, reported disease rates were generally lower (Asthma indicator, p. 5-55; Cardiovascular Disease indicator, p. 5-48; Chronic Obstructive Pulmonary Disease indicator, p. 5-52) or comparable (Preterm Delivery indicator, p. 5-67; Low Birthweight indicator, p. 5-65) in Hispanic versus non-Hispanic populations.

Limitations, Gaps, and Challenges

In answering this question, EPA reviewed general trends in morbidity and mortality of several diseases that may be related, at least in part, to contaminants in the environment to which people may be exposed. The indicators presented in this section provide an overall picture of specific disease rates or occurrence across the nation, including among some population subgroups. ROE indicator data sets, however, do not enable extensive analysis of disease trends within or across geographic regions, nor do they allow fully consistent reporting of trends across racial and ethnic groups. In addition, there are other diseases or conditions of potential interest for which no national scale data are currently available, or for which the strength of associations with environmental contaminants are still being evaluated. Specific limitations, data gaps, and challenges related to answering the question on trends in disease are highlighted below.

Geographic Patterns

Mortality data sets enable some analysis at the EPA regional level, but underlying data for most ROE indicators selected to answer this question do not currently enable meaningful analysis of geographic trends across the nation. The regional analyses presented in this report for cardiovascular disease and chronic obstructive pulmonary disease mortality reveal no discernable patterns.

Other Diseases and Conditions for Which Environmental Contaminants May Be Risk Factors

Additional data are needed to prompt or enable EPA to track other diseases and conditions with potential environmental risk factors (direct or indirect), particularly those for which unexplained increases are being noted. Examples of diseases or conditions with suggestive or growing evidence that environmental contaminants are a risk factor follow. The extent to which national-level indicators meeting ROE criteria are available to track these diseases and conditions varies.

Behavioral and neurodevelopmental disorders in children continue to receive attention. These include disabilities of the functioning brain that affect a child's behavior, motor skills, memory, or ability to learn. Examples include attention-deficit/hyperactivity disorder (ADHD), dyslexia and other learning disabilities, cerebral palsy, mental retardation, and autism. Considerable evidence exists that lead and methylmercury are associated with mental retardation and impairment of mental function and attention.[60] While the role of other

[57] Centers for Disease Control and Prevention. 2007. Summary of notifiable diseases—United States, 2005. MMWR 54(53):9. <http://www.cdc.gov/mmwr/PDF/wk/mm5453.pdf>

[58] Ibid.

[59] Akinbami, L.J. 2006. The state of childhood asthma, United States, 1980-2005. Advance data from vital and health statistics. Number 381. Hyattsville, MD: National Center for Health Statistics. <http://www.cdc.gov/nchs/data/ad/ad381.pdf>

[60] Mendola, P., S.G. Selevan, S. Gutter, and D. Rice. 2002. Environmental factors associated with a spectrum of neurodevelopmental deficits. Ment. Retard. Dev. Disabil. Res. Rev. 8(3):188-197.

environmental contaminants in contributing to some of these disorders is not fully known or understood (e.g., for ADHD), the weight of evidence suggesting relationships between behavioral and neurodevelopmental effects from exposure to polychlorinated biphenyls (PCBs), environmental tobacco smoke, and other contaminants continues to grow.[61,62] The National Health Interview Survey (NHIS) tracks ADHD and mental retardation, though the accurate reporting of these types of disorders is complicated by difficulties in diagnoses and possible underreporting (e.g., institutionalized children are excluded from the NHIS survey population).

As the U.S. population continues to age, more individuals are afflicted with neurodegenerative disorders such as Parkinson's disease and Alzheimer's disease. For example, Alzheimer's disease is the seventh leading cause of death in the nation (General Mortality indicator, p. 5-33). Such diseases are characterized by the progressive loss of neural cells, which lead to central nervous system dysfunction (e.g., memory loss, cognitive deficits, personality changes, motor control abnormalities). The etiology of these disorders is multifactorial, but in many cases the etiology is unknown. Ongoing research is exploring the role, if any, of environmental contaminant exposure (e.g., heavy metals, pesticides). Thus far, findings are largely inconclusive due to conflicting results.[63]

Diabetes was reported as the sixth leading cause of death in the U.S. in 2004 (General Mortality indicator, p. 5-33). Two types of diabetes exist. Diabetes mellitus (type 2), the most common form, is characterized by the body's resistance to insulin action and a relative deficiency of insulin. Known risk factors for diabetes mellitus include factors such as age, obesity, family history, physical inactivity, and dietary glycemic load. Type 1 diabetes results from decreased insulin production by the pancreas as part of an autoimmune response. Onset typically occurs before adulthood and believed to be triggered by genetic predisposition and possible environmental factors. Diabetes itself is a risk factor for the development of many other acute and chronic conditions. Epidemiological research has been conducted to evaluate possible associations between environmental contaminant exposure and diabetes; however, findings are inconclusive. Occupational and environmental exposures to contaminants such as arsenic, PCBs, dioxins, and nitrates have been examined.[64,65] Other endocrine and metabolic disorders, such as thyroid disorders, continue to be studied. Research continues to evaluate the extent to which various environmental contaminants are capable of disrupting endocrine function in humans (e.g., phthalates, persistent organic pollutants).

Reproductive function is another condition of interest to EPA. Scientists are studying whether environmental contaminants may cause alterations in reproductive function and contribute to conditions such as ovarian failure, decreased sperm counts, infertility, sub-fecundity, and possibly early onset of puberty. For example, components of cigarette smoke and other environmental contaminants have been studied in association with possible effects on female reproductive function.[66] Other contaminants under study include pesticides, dioxins, various metals, and solvents.

Renal disease is of interest because of the vital function of the kidneys in maintaining human health and the range of complex factors that lead to kidney dysfunction and disease. The kidneys can be seriously affected by a number of primary diseases such as hypertension and diabetes. Nephritis and nephritic syndrome were reported as the ninth leading cause of death in 2004 (General Mortality indicator, p. 5-33). EPA is interested because the kidney is known to be the target of some environmental contaminants. For example, as evidenced through occupational exposure, poisoning, and other experimental studies, exposure to heavy metals such as lead, cadmium, and mercury has been shown to be nephrotoxic.[67,68] The U.S. Renal Data System is a national data system that collects, analyzes, and distributes morbidity and mortality information about end-stage renal disease in the U.S.

Infectious diseases represent a continuing threat in the U.S. and worldwide. CDC continues to monitor infectious diseases and implement preventive strategies for infectious diseases whose incidence has increased within the past two decades or threatens to increase in the near future.[69] Infectious diseases of EPA interest may shift over time, making tracking of these diseases more of a challenge. An area of research interest for arthropod-borne diseases, and a potential issue for zoonotic diseases, is whether their incidence may change with changes in environmental condition such as land use, local weather conditions, or other environmental disturbances.

Other Data Collection Systems

To better answer the question, expanded national-level health data collection systems are needed, as well as integration of systems that collect health data. For example, the birth certificate data currently used to track birth defects on a national level have limitations (see Birth Defects indicator, p. 5-62).

[61] Schantz, S.L., J.J. Widholm, and D.C. Rice. 2003. Effects of PCB exposure on neuropsychological function in children. Review. Environ. Health Perspect. 111(3):357-376.

[62] State of California. 2005. Proposed identification of environmental tobacco smoke as a toxic air contaminant. Part B: Health effects assessment for environmental tobacco smoke. As approved by the Scientific Review Panel on June 24, 2005. California Environmental Protection Agency, Office of Environmental Health Hazard Assessment. <http://www.arb.ca.gov/regact/ets2006/ets2006.htm>

[63] Brown, R.C., A.H. Lockwood, and B.R. Sonawane. 2005. Neurodegenerative disorders: An overview of environmental risk factors. Environ. Health Perspect. 113(9):1250-1256.

[64] Longnecker, M.P., and J.L. Daniels. 2001. Environmental contaminants as etiologic factors for diabetes. Environ. Health Perspect. 109(Suppl 6):871-876.

[65] Remillard, R.B., and N.J. Bunce. 2002. Linking dioxins to diabetes: Epidemiology and biologic plausibility. Review. Environ. Health Perspect. 110(9):853-858.

[66] Mlynarcikova, A., M. Fickova, and S. Scsukova. 2005. Ovarian intrafollicular processes as a target for cigarette smoke components and selected environmental reproductive disruptors. Review. Endocr. Regul. 39(1):21-32.

[67] Klaassen, C.D., ed. 2001. Casarett and Doull's toxicology: The basic science of poisons. Sixth edition. New York, NY: McGraw-Hill.

[68] Jarup, L. 2003. Hazards of heavy metal contamination. Review. Br. Med. Bull. 68:167-182.

[69] Centers for Disease Control and Prevention. 1998. Preventing emerging diseases. A strategy for the 21st century. Atlanta, GA: U.S. Department of Health and Human Services.

CDC recognizes the need for continuing efforts to improve birth defects surveillance, and recently released improved national prevalence estimates for major birth defects looking at data reported through the National Birth Defects Prevention Network.[70] Also, as noted above, systems do not exist at the state or national level to track many of the diseases or conditions that may be related to environmental hazards. Existing environmental hazard, exposure, and disease tracking systems are not linked together.

Some efforts are underway to begin tracking exposure and health outcomes together. For example, CDC's "environmental public health tracking network" involves the collection and integration of data from environmental hazard monitoring and from human exposure and health outcome surveillance; CDC's goal is to build a national tracking network (http://www.cdc.gov/nceh/tracking/). In addition, CDC has initiated the "environmental public health indicator project," which identifies indicators of environmental hazards and health effects that state health departments can use to develop comprehensive environmental public health programs (http://www.cdc.gov/nceh/indicators/default.htm). Such programs will help bridge some existing gaps in knowledge between disease trends and environmental condition. These efforts also will enhance data collection efforts at the community level (state and local) and help ensure better temporal and spatial congruence between environmental, surveillance, and biomonitoring programs.

Lastly, data collection systems that collect data at different scales are available that may support future trend analysis. For example, CDC and the National Cancer Institute (NCI) have been combining forces to build a database of U.S. cancer statistics with data from CDC's National Program of Cancer Registries and NCI's Surveillance, Epidemiology, and End Results Program (http://apps.nccd.cdc.gov/uscs/). Cancer incidence data are available for 47 states, including six metropolitan areas, and the District of Columbia, and represent approximately 96 percent of the U.S. population.[71] Another example is asthma estimate data from CDC's state-based Behavioral Risk Factor Surveillance System.

[70] Centers for Disease Control and Prevention. 2006. Improved national prevalence estimates for 18 selected major birth defects—United States, 1999-2001. MMWR 54(51&52):1301-1305.

[71] Centers for Disease Control and Prevention and National Cancer Institute. 2006. United States cancer statistics: 2003 incidence and mortality. U.S. Cancer Statistics Working Group. <http://www.cdc.gov/cancer/npcr/npcrpdfs/US_Cancer_Statistics_2003_Incidence_and_Mortality.pdf>